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CASES OF RECOVERY FROM SYMPTOMS POINTING TO THE PRESENCE OF PROGRESSIVE ORGANIC CEREBRAL DISEASE.

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INTERESTING as all cases of intracranial disease are from many points of view, those are specially interesting to the practical physician in which recovery ensues from symptoms which former experience had led him to regard as indicative of progressive mischief tending to a fatal result. Of recovery, more or less complete, from all kinds of so-called "functional" disorders we have, of course, ample experience. Of recovery also more or less complete, from small hæmorrhagic effusions or from small patches of softening, again clear evidence is sufficiently abundant. Simple inflammation of the surface, or even of the substance, of the brain, no doubt subsides, leaving little or no clinical trace of its pre-existence; and, probably, this is of more common occurrence than most of us suspect. Syphilitic affections, again, are not unfrequently benefited, and sometimes cured, by appropriate treatment. Nevertheless it cannot be denied that symptoms indicative of obvious inflammation of the cerebral meninges, symptoms pointing to the presence of cerebral tubercle, symptoms characteristic of tumours of the brain, and even symptoms apparently referrible to progressive degenerative processes,

are properly regarded as of the gravest omen, and in the great majority of cases foretell a fatal issue.

The cases which I am about to narrate derive their chief interest from such considerations as have inspired the foregoing paragraph. Two of them impressed me greatly; because from their symptoms and progress I had gradually been confirmed in the belief that they were hopeless, when lo! amendment took place, and before long the patients were restored to health.

The first case was that of a young woman, who, when she came under my care, had been complaining for three weeks of abdominal pain; who on her admission into hospital was suffering from subacute peritonitis, with fluid effusion into the abdominal cavity; and who during the next seven weeks presented symptoms which were ascribed, and I think rightly ascribed, to tubercular peritonitis. She had general pain and tenderness of the belly, with more or less fulness, constant sickness, hectic temperature, progressive emaciation and debility, which appeared to be uninfluenced beneficially by any kind of treatment that was adopted. Then she began to squint, from partial paralysis of both external recti, and of the left internal rectus, to present oscillation of the eyeballs, to lose memory, and to have delusions. It was naturally, and I still think correctly, surmised that intracranial tuberculosis had been added to the abdominal tuberculosis; it was also, I think, naturally surmised that the condition of the patient, previously sufficiently grave, had now become hopeless. In the course of a few days, however, to my surprise, the aspect of affairs began to brighten. By degrees, yet rapidly, the temperature became normal, the abdominal symptoms subsided, the sickness ceased, appetite returned, and the patient began to sit up and take interest in all that was going on around her. A little later her squint also disappeared, and she lost her delusions; and six or seven weeks after the first appearance of squint, she left St. Thomas's for a convalescent hospital, still forgetful and still presenting slight nystagmus, but in all other respects perfectly restored to health. I saw her on February 3rd of the present year; she came to see me at my request. I found that for the last twelve

months she had been working at a laundry, that she had had good health, and no recurrence of her former symptoms, and that she looked well, but that she had never recovered her previous good memory. The following is a detailed account of this case:—

CASE I.—*Tubercular peritonitis* (?)—*Tubercular meningitis, or tubercular tumour of the brain* (?)—*Recovery.*

ARABELLA D., an unmarried woman 23 years of age, was admitted under my care on the 4th of October, 1883. Her health had been good up to three weeks ago, when, as she was carrying a heavy tray, she felt a sudden sharp pain in the umbilical region. She has suffered from abdominal pain of varying severity ever since; and her abdomen has enlarged.

She is a dark-complexioned, good-looking, fairly nourished girl, complaining of abdominal swelling, and of pain referred chiefly to the hypochondriac regions. The belly is uniformly distended, and measures 35 inches in girth at the umbilicus. It is resonant in front, and dull in the flanks, as she lies on her back; and the relations of the resonant and dull areæ vary with position. There is some tenderness on pressure; but no evidence of tumour, or of enlargement of liver or spleen. No anasarca in legs or elsewhere. The thoracic organs are normal, excepting that the heart's apex beats a little higher than natural, and that there is some crepitation at the bases of the lungs. There is no evidence of mischief at the apices. Appetite bad; tongue furred, but moist; pulse 80; respirations 20; temperature varying from 98·4 to 99. Urine, sp. g. 1024; no albumen.

During the next week there was little change in her condition, excepting that the ascites gradually increased, until the girth of the abdomen measured more than 40 inches, and resonance disappeared excepting on deep pressure. The impairment of appetite continued; she complained constantly of pain and tenderness in the abdomen, which were not always referred, as at first, to the hypochondriac regions; her temperature rose daily above 100, but varied between 98·4 and 100·6; and her urine was of high specific gravity, and contained abundant urates.

On the 11th she had an attack of diarrhœa, and her bowels were moved eight times; she vomited a good deal, complained of much epigastric pain, and her temperature rose in the morning to 102·2. The tongue was covered with brown fur.

The diarrhœa continued off and on for a week; during the first

two or three days of which she still vomited. She complained during the week of pain and tenderness of the abdomen, chiefly in the epigastric region, and also of pain between the shoulders; her appetite was very bad, her tongue brown and inclined to be dry, and her urine presented a little albumen and a few granular and hyaline casts. The temperature reached 102·8 on the 12th, and subsequently varied between 98·4 and 101. The ascites remained unaltered.

From the end of this week to about the 22nd of November, the patient got steadily weaker and thinner, and suffered from profuse nocturnal perspirations. Her bowels were variable, but on the whole inclined to constipation. She suffered from sickness, and often vomited several times in the day. Her tongue was coated; her appetite very bad. The pain and tenderness in the abdomen never left her; and at times the pain, which was paroxysmal and referred mainly to the umbilical and epigastric regions, was very severe. But during the last fortnight of the time the ascitic fluid disappeared, and the girth of the abdomen became reduced to 27½ inches. The reduction, however, was not attended with any diminution of pain or tenderness, and the abdominal walls became rigid. The temperature varied for the most part between 99 and 101, occasionally becoming sub-normal, and at times rising above 101. From the 13th of November onwards, however, it never reached 100, and was usually normal or sub-normal. The pulse, which was always feeble, ranged from 80 to 116. The urine continued scanty and high-coloured, and usually contained a trace of albumen. The catamenia, which were due about the time of her admission, did not make their appearance either then or subsequently. She had no cough or sign of disease at the apices; but the sub-crepitation, audible at the bases on admission, was audible from time to time subsequently.

On the 24th of November it was noted that for a day or two she had been complaining of double vision; and it was found that she had an internal squint with both eyes (the left external rectus being the weaker), and nystagmus. But she had no headache, giddiness, tremors, or colour-blindness.

On the 26th she was examined by the ophthalmic surgeon, who observed that there was defective movement of both eyes outwards, and slight impairment of inward movement in the left eye, and that the internal structures of the eyes were healthy. There was still nystagmus. Still also she complained of dull aching pain across the upper part of the abdomen; she vomited a little at night; and the urine contained a trace of albumen. Her temperature was normal.

28*th*.—The condition of the eyes is unaltered; and there is still some pain across the epigastrium. But the patient has been improving in all other respects during the last few days; she is bright and cheerful, and hungry; sickness has ceased; and her abdominal uneasiness has so much diminished that she has been able to sleep without the hypodermic injections of morphia, to which she has been accustomed almost ever since admission. Pulse 114; temperature normal; urine 1030, many oxalate of lime crystals, and a trace of albumen. Ever since the eyes have become affected the patient has suffered from loss of memory (cannot recollect the days of the week, the times of my visits, whether she has had her dinner, &c.) and from delusions (such as that her mother has brought her a pineapple; that her father, who has long been dead, has been sitting beside her).

From this time forwards there was progressive improvement. The patient had no return of sickness, and enjoyed good appetite; her abdomen became more and more flaccid, and free from pain, which finally disappeared wholly; the diplopia gradually diminished, and had quite subsided by the 16th of December, but nystagmus (especially when she looked to the extreme left) continued; her temperature ranged from about 97 to 98·2; she put on flesh, recovered strength, and became lively and happy. On the 2nd of December, and for a few days subsequently, she complained of pain across the forehead, and slight giddiness; but these symptoms did not recur. The fundi of the eyes, before she left the hospital, were carefully examined by Mr. Nettleship, who failed to discover any choroidal tubercles, and reported them as being quite healthy.

On the 9th of January she was sent to a convalescent hospital. At that date she expressed herself as feeling, and looked, quite well; she was enjoying her food, and helping in the ward; the abdomen was soft and void of pain, tenderness, or tumour; the urine was free from albumen; she had no headache or double vision; but slight nystagmus was still observable when she looked out of the corners of her eyes; and she was still forgetful. There was no evidence of pulmonary disease. She weighed 6 stone 11 lb.

During her stay in the hospital she was treated mainly with morphia, administered subcutaneously, and tonics.

There is, no doubt, much that is difficult to understand in the narrative which has just been given. It is especially difficult to comprehend how or why the abdominal symptoms should have subsided just as the cerebral symptoms came on, and how or why the latter should in their turn have passed

away, leaving the patient apparently healthy. I think that no one who watched the case during life doubted, and that few of those who read the notes carefully can doubt, that the girl was suffering from tubercular peritonitis. The symptoms and progress of the case were exactly what one constantly witnesses in that disease, and, so far as I know, in no other. Moreover recovery from the symptoms of tubercular-peritonitis, though certainly rare, is not unprecedented. But if she had tubercular peritonitis, it is difficult to believe that her cerebral symptoms could have been due to anything else than intracranial tuberculosis. I do not see how the combination of double internal squint, nystagmus and loss of memory, could have been merely functional. There was no ground whatever, from the antecedents of the girl, to suspect syphilis; moreover she recovered without the use of antisyphilitic treatment. No doubt inflammation at the base of the brain may be idiopathic; and this might furnish the explanation of her symptoms. But the arguments which might be adduced against the presence of simple meningitis are of the same kind as those that might be adduced against the presence of tubercular meningitis; and it is the less likely of the two explanations in this case, inasmuch as there was strong evidence in favour of the presence of tubercles elsewhere. I believe that the patient was suffering from cerebral tuberculosis; but whether this was in the form of slight basal meningitis, or of tumours in the substance of the cerebellum or elsewhere, I cannot venture to decide. The absence of optic neuritis, which is not uncommon in either of these affections, does not help the diagnosis.

In connection with this case I may briefly advert to another which came under my notice some years ago, and which has some kind of relation to it.

Case II.

On the 5th of May, 1875, a servant-girl, 15 years of age, came under my care. She had had good health up to three weeks before I saw her. Then she began to suffer from pains in the head, back and abdomen, giddiness, drowsiness by day and restlessness at night. Also she began to see double.

On admission, she was a fairly healthy-looking girl, complaining

of headache, giddiness and double vision. She was very drowsy, and indisposed to answer or take notice. Her pulse was 48, her respirations 24, her temperature normal. She had paralysis of both external recti, and well-marked double optic neuritis of recent origin. For some days afterwards she remained somewhat drowsy and torpid, with slow pulse, and at times irregular respiration of the Cheyne-Stokes character; her temperature at the same time being slightly below the normal. Then she gradually improved, her squint disappeared, and at the end of four weeks from her admission left the hospital well, excepting for the persistence of optic neuritis.

She was re-admitted twelve days later, suffering from weakness, pains in back, short breath on exertion, and slight cough. She also complained of a little pain in the right temple, weakness of eyes, and some giddiness when walking. She was suffering from slight bronchial catarrh, and while in the hospital had an attack of erythema nodosum, during which her temperature varied between 99 and 102, and on one occasion rose to 102·8. She remained in the hospital two months, and left fairly well. On leaving, there was still marked evidence of double optic neuritis but no other definite indications of cerebral disease. There was no clear indication of tuberculosis. But I thought at the time, and am still inclined to think, that her symptoms were due to tubercular meningitis.

The third case was that of a woman, 39 years of age, who in the midst of apparently good health was attacked with double vision and giddiness, followed in about three weeks by headache and inability to stand. Then she vomited, and complained of numbness of the right half of the upper lip. At the end of a month she had headache, staggered like a tipsy person when she had attempted to walk, suffered from distressing nausea, had incomplete paralysis of the right portio dura, paralysis of the right external rectus, and slight paralysis of the left internal rectus, horizontal nystagmus when she turned her eyes strongly to the left, and contraction of the right half of the field of vision. She had no other paralysis, no impairment of sensation, no affection of the internal muscles of the eyes, no optic neuritis, no colour-blindness, and no loss of taste or smell. Soon afterwards the left external rectus became paralysed. Then came numbness and tingling in the feet, with involuntary jumpings of the legs, tendency to

stiffness, and increase of tendon reflexes. A short time afterwards a little twitching of the left angle of the mouth was observed, which soon extended to the left eyelids and to the left hand. By this time too she had become colour-blind with both eyes. Lastly, numbness and weakness of the right arm supervened, followed by marked contracture. At the end of three months from the beginning of her illness, her symptoms were at their worst; and, looking to their gravity and their rapid progress, I must confess that I took a very unfavourable view of the patient's chances of recovery. Nevertheless amendment took place; one by one her symptoms disappeared; and in two months she left the hospital in all essential points restored to health. And now at the end of nearly two years she is, I believe, earning her livelihood as a nurse. The following is a full account of this case.

CASE III.—*Symptoms pointing to progressive disease in the neighbourhood of the fourth ventricle, coming on gradually, and finally subsiding under treatment.*

ELIZA N., a nurse, single, aged 39, was admitted into St. Thomas's under my care on the 17th of January, 1883.

Excepting that she had had an attack of enteric fever ten years previously, and subsequently occasional slight rheumatic pains, she had enjoyed excellent health, until her present illness began. There was no history or evidence of syphilis.

She had been attending on a private paralytic patient, when suddenly a month ago, while feeding him, she was attacked with double vision. This was accompanied by a sense of giddiness and nausea—a feeling of sea-sickness, as she termed it. These symptoms continued, and about three weeks afterwards she found on getting out of bed that she could not stand, and in fact tumbled while stooping for her slippers. On the 12th she first complained of headache over both the frontal and the occipital regions, a sense of pressure, and as if her head were too heavy for her. On the 15th she vomited, and noticed numbness and weakness of right half of upper lip.

She is a healthy-looking, well-nourished woman, complaining of headache, nausea, giddiness, and consequent inability to stand, and difficulty in using the upper lip on the right side. Her headache is severe and more or less general, but is referred mainly to the frontal region. There is an area of tenderness to percussion,

however, at the back of the left parietal bone. The forehead also is somewhat tender to percussion. The nausea is distressing, but is present mainly when she sits up in bed, or tries to stand. She is unable to walk without assistance, or even to stand; staggers like a drunken person, and has a tendency (she says) to fall over to the left rather than to the right side. There are no ataxic movements. There is slight, but obvious, paralysis of the right facial nerve; the right eyelids close imperfectly; the right upper lip evidently acts feebly; and the right side of the face generally is smoother than the other. Nevertheless, the right angle of the mouth moves freely when she laughs. The tongue is protruded slightly towards the left, but when the organ is drawn in again its left side looks plumper and lies higher than the right. There is no obvious difference in the appearance or action of the two sides of the soft palate; but the uvula is concave towards the right side, and its apex points in that direction. She presents a marked squint. The right external rectus appears to be completely paralysed; and the left internal rectus slightly paralysed; and there is well-marked horizontal nystagmus when she looks strongly to the left. Pupils normal. There is no optic neuritis. She can distinguish the forms of objects and colours perfectly; but there is apparently some contraction of the field of vision towards the right side. Smell and taste and speech are unimpaired. No affection of ears, excepting that she has slight deafness on the left side, which dates from childhood. No paralysis or anæsthesia of limbs; tendon and superficial reflexes normal; mental condition healthy; no hysterical symptoms. Thoracic and abdominal organs healthy; tongue clean; appetite fair; bowels open; catamenia regular; urine normal; temperature 99·6.

The presence of the symptoms above detailed were confirmed during the next few days by repeated and careful examination. And Mr. Nettleship not only concurred in the description of the eyes above given, but, by investigation of the field of vision, discovered that the right half was so largely contracted for both eyes, as almost to cause hemiopia. Her headache varied in severity, but was rarely if ever absent, and often intense. Her nausea remained for the most part in abeyance so long as she lay perfectly still, but became severe whenever she sat up in bed, and especially when she was made to stand. Her appetite was maintained. She continued quite unable to stand alone.

On the 23rd it was noticed that there was decided weakness of the left external rectus, in addition to the former ocular defects. This became more pronounced during the next four days.

On the 27th she first experienced a feeling of numbness and

coldness at the bottom of the feet, which in the course of a few days amounted to a sense of pins and needles. On the 12th of February she complained that her legs felt stiff, and that they jumped at times. She could move her legs pretty freely; but they tended to become rigid, especially at her knee- and ankle-joints; there was marked exaggeration of the tendon reflexes, and on the left side ankle-clonus. These phenomena continued for a time, the left leg being worse than the other. Ankle-clonus was obtained later on the right side, which also was somewhat more numb than the other.

On the 3rd of February it was observed that the facial paralysis had increased; although when laughing the right angle of the mouth was still drawn up, at least as much as the left; but it was also noticed that there were frequent twitchings of the left angle of the mouth. These twitchings continued; and about the 20th it was noted that she had also occasional twitchings in the left eyelids, and slight tremors in the left hand.

She had complained for a short time that her eyesight was not so clear as it had been; and on the 1st of March this was again carefully tested. At that time the ocular paralyses remained; the fields of vision were as before; and the discs were quite clear, but doubtfully pale. But now she was colour-blind. She could not distinguish greens or reds, and confounded them with brown, and sometimes with grey. Bright yellow was called white. Bright blue and lilac were both called dark blue. She was sure also that her vision was in other respects worse than it had been.

On the 30th of March, after she had been suffering for a day or two from much more intense pain than usual, she for the first time complained of a feeling of pins and needles in the right hand and arm, and of pain in the right shoulder. The arm also became weak, and in the course of a week or two slightly flexed at the several joints, the fingers especially suffering; and she had more or less pain from the shoulder downwards. This paralytic affection of the arm was never complete; but attained its maximum towards the end of April.

During the greater part of the time terminating with the last date, the patient had on the whole been getting slowly but steadily worse; the pain in the head (which varied in position, but became more and more localised in the neighbourhood of the back part of the left parietal bone, as time went on) was constant, but liable to severe exacerbations; the sense of nausea on the slightest movement continued; and from time to time (as has been shown) additional paralytic phenomena arose. Nevertheless during the month of April some favourable indications were manifested. On

the 11th she saw singly for the first time since admission. And on the 15th the following note was taken: "Has not seen double since the 11th, and now the movements of the eyes appear to be perfect. She distinguishes colours better than she did a little while back; she recognises bright greens and blues, but calls red black, and yellow dirty white. There is still nystagmus when looking to the extreme left. Also there seems some improvement in the facial palsy; at any rate she closes her eye better than she did, and says that the right side of the face is less stiff. Complains that left side of head is heavier than right." From this time forward there was no return of double vision; and her power of appreciating colours was slowly restored.

Late in April, or early in May, the patient began to improve decidedly. She still suffered from intense headache, giddiness, and nausea; but the attacks were not so frequent; and intermissions occurred which became longer and longer. Her appetite was better, and she felt stronger. About the 10th or 11th of May she began to sit up for a few hours in the evening; and a week or two later got up in the afternoons, and even began to walk about with assistance. It remains only to add, that her various paralytic symptoms gradually cleared up; and that, when she left the hospital on the 4th of June, there were scarcely any traces of nausea or headache, the right facial nerve was not visibly paralysed (although she said that side of the face was still stiff, and that there was still a little difficulty in retaining fluids within the lips), the right hand and arm had almost completely recovered their power, and could be used freely, there had been no starting in the legs for some weeks, and (though the right leg was still somewhat stiff) she could walk well.

She went for a month to a convalescent hospital; at the end of which time she presented herself for examination. She then seemed quite well, and expressed herself as being able to resume her occupation. There were no discoverable signs of paralysis, and her appreciation of colours was entirely restored. I saw her some months later, and she remained well. I may add that there was still a little twitching about the left angle of the mouth. I think it probable, however, that this was an old affair.

During the greater part of the patient's illness her temperature varied between 99° and 100°; but occasionally it rose to between 101° and 102°, and more frequently descended to the normal. I cannot say that it improved materially as her condition improved in other respects.

The treatment adopted was mainly the subcutaneous injection of morphia, repeated often two or three times a day, for the relief

of headache; and the occasional use of leeches behind the ears (the application of which was usually followed by benefit), of blisters and of ice.

For the first week or two I prescribed 5 grains of iodide of potassium, and 40 minims of solution of perchloride of mercury, to be taken three times a day. Then this was replaced for a time by bromide of potassium, in 20-grain doses. Then she was treated, for reasons not referred to in the above notes, at one time with some stomachic mixture, at another time with some cough mixture. The iodide of potassium and mercury were resumed on the 15th of April, and continued until she left.

What was the matter with this patient? Had she a tumour of the brain? Many of her symptoms—her localised headache, giddiness, nausea and rapid extension of symptoms—suggested this explanation. But the absence of optic neuritis and her final recovery seemed to negative this view. Was her disease a functional one merely? I think a decided "no" may be answered to this question. There was no history of hysteria; and she was not at all emotional. Moreover the character of the symptoms and their mode of development were not in accordance with one's experience of mere functional disorder. That her symptoms could not have been due to obstruction of arteries with consequent softening of some limited tract of brain-tissue, or to hæmorrhage, is clear from the fact that their development extended over three or four months. In many respects her case presented a close analogy to cases of ophthalmoplegia, presumably due to degenerative changes, or chronic inflammatory processes. In these we not unfrequently observe headache, giddiness, sickness, and (besides the paralysis of the eye-muscles) various other paralyses, anæsthesia of limited distribution, and involvement of one or more of the special senses, without optic neuritis. But these cases, so far as I know them, are of much slower progress than hers, and do not tend to recover. I do not see, however, why such cases should not occasionally improve or even recover; and on the whole I lean to the opinion that, in this patient's case, the symptoms were really due to some subacute progressive inflammatory process taking its origin somewhere about the floor of the fourth ventricle, and spreading thence in depth and surface.

Her recovery under the use of iodide of potassium and mercury suggests a syphilitic origin to her symptoms. I never dared myself to ask her whether she had had this disease. She was a very healthy-looking woman; there was no lump or scar or stain about her body to suggest that she had ever had anything of the kind; and her demeanour was such as to disarm suspicion. Nevertheless the possibility of the affection being syphilitic cannot be altogether excluded from consideration.

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ON THE NATURE OF THE SPINAL LESION IN POLIO-MYELITIS ANTERIOR ACUTA, OR INFANTILE PARALYSIS.

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It is the opinion of well-nigh all pathologists that the paralysis and atrophy so typical of this disease are the result of destruction of the ganglionic cells in the anterior grey cornua. This is the position taken up by nearly all clinical and pathological teachers, without any fear of contradiction; and, indeed, numerous post-mortem examinations, made in cases where death has resulted from some independent affection, some time after the onset of the lesion, have proved its truth. It has, however, fallen to the lot of very few to examine the cord of a child very soon after it has become affected, in what might be called the acute or inflammatory stage, for, as is well known, it is a lesion that very rarely proves fatal; consequently the exact nature of the essential and primary lesion is little more than a matter of conjecture. Such a chance has recently occurred to me—for which opportunity I am indebted to my friend Dr. Pope, of South Shields. The child whose spinal lesion I am about to describe was a girl aged five. At breakfast-time of the day of her death she was in her usual health, but shortly after her meal she felt sick and vomited. She was then put to bed, when she slept for several hours. Early in the afternoon it was observed that she was feverish and evidently in a dying condition, an observation that was soon consummated, as her death took place six or seven hours after the commencement of her illness—the result, as was afterwards surmised, of respiratory paralysis. At Dr. Pope's request I made a post-mortem

examination. The body was well nourished. The heart was healthy. The lungs were somewhat congested, and in the left was found a small nodule of catarrhal pneumonia, about the size of a walnut. The abdominal cavity and its organs were normal. The brain appeared to be healthy in every respect. Failing to account for death by the changes noted in the lungs, which, so far, were the only morbid conditions detected, I proceeded to examine the upper part of the spinal cord, when the usual transverse sections at various levels from the medulla oblongata downwards brought to light an altered condition of the anterior grey horns between the third and fourth cervical

nerves, the appearances at the level of the latter suggesting *red softening*. This portion of the cord was hardened in a solution of bichromate of ammonium, cut with an ice-freezing microtome, and stained with carmine.

When examined by the low power of the microscope (half-inch), the extreme vascularity of the anterior horns was at once apparent. In some of the sections, in addition to the unusual display of capillaries, minute patches of yellow figured prominently upon the carmine groundwork. The ganglionic cells were seen to be numerous and distinct, though apparently swollen. With the high power ($\frac{1}{6}$ inch, see figure above), the

blood-vessels, distended with corpuscles, formed a striking feature, not only of the anterior horns, but of the anterior white columns and middle and anterior portions of the posterior horns as well, for the inflammatory mischief was by no means confined to the first-named region. Following the tracks of the arteries (internal and external anterior root arteries, and the antero-lateral branch) as they made their way from the front of the periphery of the cord, through the white matters to enter the anterior horns, minute hæmorrhages were scattered here and there, whilst congestion, with free leucocytes, was apparent. In places the neuroglia was swollen and so altered as to obliterate in part the meshy character of the structure, and thus conceal the cut ends of the axis cylinders. In this way the bulk of the anterior columns formed a marked contrast to the beautiful and distinct arrangement of neurogliar network, with the nerve fibres seen as central dots, which marked the postero-lateral columns. As the anterior grey horn was approached, the signs of inflammation increased, attaining a maximum as the swollen and somewhat ill-defined mass of grey matter was reached. Here the dilated vessels were crammed full of corpuscles, whilst the capillary network was rendered most distinct, each vessel containing its single or double row of cells. The minute hæmorrhages, which were seen as collections of yellow-coloured blood-cells, formed the most striking feature of each field that was examined. These extended as far back as the distribution of the posterior branches of the central artery on each side, that is, quite into the posterior horns, but they were most numerous in the anterior cornu, about the ganglionic cells. The large cells formed prominent objects, though they were apparently swollen, granular, and rather ill-defined; and it was observed that the majority had lost their nuclei, though here and there a ganglionic cell with a nucleus could be distinguished. Their processes were no longer visible, and they appeared to fill completely the spaces in which they lay. The cells that presented the most obvious changes were more or less surrounded with dilated and blocked capillaries and minute hæmorrhages; some, indeed, appeared to be imbedded in blood-corpuscles.

In most of the sections it was apparent that one horn had suffered more than its fellow, though in most of those examined both horns showed inflammatory changes. The grey fibres which form the bulk of the anterior horns were swollen and obscure, the whole presenting an indefinite and somewhat granular appearance, which contrasted strangely with the uninjured portions of the posterior horns and roots.

The pronounced changes above described were only observed in a very limited portion of the spinal cord, for as the sections gradually ascended or descended from the affected area, a return to the normal state was brought to light, the signs of inflammation diminishing in intensity, until a few isolated hæmorrhages with distended capillaries alone were found.

Although the accident of the situation of the lesion, attacking as it did the region of the origin of the phrenic nerve, prevented the little patient from living long enough after the onset of the inflammation to admit of an exact clinical diagnosis, I do not imagine that any one will be found to question the identity of her case with *polio-myelitis anterior acuta*, and I make no apology for the assumption that they are the same. I think, therefore, that the case is one of considerable importance, as demonstrating the early changes that lead up to the later degenerations which all are so familiar with in old-standing cases of infantile paralysis.

In the year 1870, Dr. Clifford Allbutt recorded in the 'Lançet'[1] a case which has been frequently quoted by writers on infantile paralysis (Ross and others), and I think erroneously, as an example of the disease. In this case an examination of the cord, soon after the occurrence of the lesion, revealed two hæmorrhages in the cervical region, "one a small one in the left posterior horn, and the other larger, in the right posterior horn, breaking into the lateral column." I do not think this case can be fairly regarded as a true example of *infantile spinal palsy*, as Dr. Allbutt points out that the paralysis, which affected all four extremities, came on a few minutes after the child, whose age was six months, had been lifted sharply, when its head fell heavily forward. Obviously there is here an element of traumatism foreign to most cases of acute anterior polio-

[1] 'Lancet,' 1870, vol. ii. p. 84.

myelitis of infants; and, further, the hæmorrhages appeared to be confined to the posterior half of the cord, a region which cannot be looked upon as the real seat of the disease, though affected in the case I have described, and also in one to which I am about to refer, recorded by Dr. Angel Money. I am therefore disposed, with due deference to Dr. Allbutt and those who, in quoting the record, have shared his opinion, to think that the hæmorrhages in the case were the result of injury rather than inflammation.

In the 'Transactions of the Pathological Society of London' for 1884 (just published), page 45, Dr. Angel Money describes under the title, "The Spinal Cord of a recent and old case of Infantile Palsy," the appearances of the cord of a child, aged two years, who died of pulmonary disease sixteen weeks after the onset of an attack of acute anterior polio-myelitis. The changes found in the lumbar enlargement were "(1) great distension and thrombosis of vessels, especially in the anterior cornua; (2) infiltration of the cornua with abundant leucocytes; (3) absence of large multipolar or other nerve cells." Dr. Angel Money goes on to state as follows:—"Further, it was seen that the disease was not confined to the anterior horns, but spread forwards, outwards, and backwards; though the principal focus of the mischief was certainly the centre of the anterior horn." This observation, published some time after I had written the above short account of the spinal cord in Dr. Pope's case, confirms my own, in so far as it shows that the inflammation is not by any means confined to the large ganglionic cells, or even to the anterior horns; but the fact that it was made sixteen weeks subsequent to the attack, is sufficient to explain the apparent slight differences between Dr. Money's and my own description of the primary lesion.

Althaus states in his essay,[1] and I believe him to be correct, that there were at that date no *post-mortem* records where the child had not been paralysed for at least two months, nor am I aware that any more recent examinations have been made since he wrote. The case I have recorded would appear to set at rest the question discussed by Charcot, Roth, Dujardin, and others, namely, whether the inflammation is

[1] 'On Infantile Paralysis.' London, Longmans, 1878.

parenchymatous or interstitial, for it is obvious that the several parts affected—the neuroglia, the nerve fibres, and the ganglionic cells—must take part, though they may not all share alike, in the destructive processes induced by the inflammation. Indeed, the brunt would appear to fall on the large cells, a fact revealed alike by the examinations of old-standing cases, and the clinical features of the disease.

Reference has already been made to the extension of the inflammation beyond the limits prescribed by the various writers on the subject, for it will be recollected that though the fresh changes were found most abundantly in the anterior horns, they, nevertheless, affected the antero-lateral white columns, and part of the posterior horns as well; in fact, the anterior cornua were only the centres of inflamed areas, which reached considerably beyond their limits. It will, therefore, become a question whether the name proposed by Kussmaul, and adopted by the majority of writers—anterior polio-myelitis—is really an appropriate one, though usage in the nomenclature of disease, as in other matters, will be found to resent interference.

In conclusion, I would like to point out two ways in which the recognition of the real nature of the primary lesion of infantile paralysis is of importance. In the first place, it throws light upon the attacks of *temporary spinal paralysis of infants*—seizures, if I may be allowed the expression, which I confess have puzzled me not a little. An example, briefly related, may not be out of place. A little boy, aged 16 months, whilst cutting his eye-teeth, was observed one day to be unusually peevish when being dressed to go into the garden in his perambulator. As he continued to fret, his nurse attempted to amuse him by offering him a ball, when she discovered that he could not raise the left arm to seize it. He was at once taken into the house and undressed, in order to discover if the arm had been injured, when it was found that he was feverish, and that the left arm was very hot and painful (hyperæsthetic). There was no bruising or other evidence of injury, but the limb hung down completely paralysed. When asked to clap his hands, an accomplishment which he had acquired some time before, he contented

himself with slapping his right hand against his leg, ignoring the left entirely. The paralysis lasted for 24 hours, and recovery of motion, which was apparently accelerated by the application of cold to the limb, was first observed in the fingers.

It will not be difficult to explain such an attack by the supposition that a limited area of the left anterior horn in the cervical enlargement had suddenly become congested, the lesion stopping short of actual inflammation.

In the next place, the necessity for early energetic anti-phlogistic treatment is emphasised, in order to overcome the inflammation, or reduce it as much as possible, by rest, cold to the spine and affected limb, leeches, or, as recommended by Althaus, the hypodermic injection of ergotin; and the fact that some attacks are only temporary, whilst in others, and indeed nearly all, there is a tendency to recovery shown by some of the muscles at first paralysed, should stimulate us to endeavour to recognise the condition as early as possible, and to carry out thoroughly the curative means at our disposal.

ON SOME CENTRAL AFFECTIONS OF VISION.

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THE labours of Ferrier, Munk, Goltz, and others have done much during the last few years to advance our knowledge of the sensory functions of the brain, especially of the cerebrum. They have used the experimental method, and their conclusions apply primarily not to man, but to the monkey, dog, and other lower animals.

The spectacle of doctors disagreeing is one that many people can observe with equanimity, and some few, unfortunately, with unaffected pleasure, especially when the doctors are vivisectionists, and the moral can be so easily pointed. In this question of the functions of the cerebrum, the results are no doubt differently interpreted by Ferrier and Goltz. It would be strange if there were not differences in so complicated a problem as that of the functions of the most highly organised of matter. But putting aside the rival interpretations, there still remains to us the solid store of facts which these observers have accumulated, and the influence of which is already perceptible in the fresh impetus given to the clinical and pathological investigation of the functions of the human brain. It is to this clinico-pathological aspect of the question that we propose to direct attention in this paper.

A few years ago Ferrier, searching the records of cerebral disease for mention of affections of sight, hearing, and the other senses, met with such poor success, that he concluded the latency was not a latency of symptoms but of observation. The justness of this criticism is indisputable, and the recent progress of this department of medicine has amply vindicated it. To take the affections of sight as an example; the last

decade has seen marked advance in our knowledge of amblyopia, hemianopsia, "psychical blindness," and word-blindness; and we find that in certain cerebral diseases affections of sight instead of being rare occurrences, are as frequent as the occurrence of aphasia.

There are many ways in which the problem of the functions of the brain and their localisation may be approached by the physician. We shall limit ourselves here to the consideration of sight and its connections; and we shall find it convenient to divide the subject into two parts; the first treating of sight and its associations, or, to adopt a more physiological phrase, visual reflexes, using the word reflex in a very broad and perhaps strained sense; the second, treating of the localisation of these reflexes in the brain.

Visual Reflexes.

Much confusion has arisen from the introduction of the terms of a subjective psychology into the study of objective brain physiology. Writers have puzzled their readers, and often we fear themselves, in their attempts to define consciousness, and distinguish between sensation, perception, apperception, cognition, and the like. These abstract distinctions have their value and their place, but we must not forget that they are merely stations that a subjective psychology has made, not found, on the unbroken line of a continuous nervous process. It will be our endeavour to banish these terms as far as possible from our inquiry, and to proceed as objectively as we can.

Stated briefly, our aim is to note the effect produced on a person by the sight of different objects. It may be that light, and light alone, has influence on him, and that the contraction of the pupil is the only reflex that can be roused; or he may notice persons and things, and follow them blankly with his eyes; or he may grasp at objects which are offered him, and yet not know what they are, or how to use them. A still higher development may be reached, and the sight of food may suggest the movements requisite for eating; well-known faces may be recognised, writing understood, or the most complex visual pictures and symbols appreciated and acted on.

It is of course absurd to think of enumerating all the visual reflexes that are to be found even in a man of the meanest intellectual endowments. The most laborious lexicographer would shrink from the task of compiling a table of contents of even this one department of the human mind, though there is certainly a limit to its contents. We can, however, divide the visual reflexes into certain great classes, and arrange them in an order advancing from the simple to the complex, from the completely organised to the least organised, from the stable to the unstable, preserving, in fact, as far as possible, the order of their evolution, the inverse of which is the well-known order of dissolution or regression upon which Herbert Spencer and Ribot lay such stress in their writings.

Such a classification will demand a wide and careful investigation. For this purpose the dawn of the visual reflexes must be observed in infants, and such observations as Darwin's and Preyer's pressed into service. In the same way the gradual lapsing of the reflexes in the decay of age will be of use. The phenomena of sleep, somnambulism, mesmerism, automatism, will all yield valuable information, which can be supplemented by an examination of the state of the reflexes in the different stages of the action of anæsthetics, and of toxic agents such as alcohol. Then, too, the field of observation must include the visual reflexes in idiocy and imbecility; in the peculiar mental states that follow epileptic and paralytic attacks; in word-blindness and the allied affections; and in that most complicated group of all, insanity.

One of the chief difficulties in satisfactorily grouping together the visual reflexes, and arranging the classes in an order advancing from the simple to the complex, lies in the fact that the simplest and most direct reflexes are, by the clustering of associations around them, gradually raised into the position of complicated and involved reflexes. The retino-muscular reflexes, for instance, that are concerned in focal and axial adjustments, and that are associated with the idea of distance, are among the simplest of the reflexes, and are developed in our earliest childhood. For example, a child soon learns only to grasp at what is approximately within reach. But these same reflexes in the mariner, the sportsman, the land-

surveyor, are by a process of education associated with much juster ideas of distance, and their suggestions are checked by a variety of other recollections. Thus, although these retino-muscular reflexes are the basis of all ideas of distance, yet it would not be a natural classification if we regarded all judgments of distance as contained within this elementary group. To take another example: there are certain protective visual reflexes that form a very convenient clinical group. In this group we include such acts as blinking, when a finger is thrust up to the eye; starting, if a sudden stabbing movement is made with a knife; aversion, if a red-hot poker is offered, or if a cup of steaming water is held to be drunk out of. These reflexes are based on some of our earliest and most powerful impressions, and they constitute a group of what may be called low and well-organised reflexes. But it is difficult to formulate an accurate definition of the group, for it gradually shades off into higher reflexes, based on less frequent or less powerful impressions, and exhibiting greater intellectual action. This is a difficulty that is felt in all the groups, but it must not discourage; it is felt in all classifications, for nature abhors sharp distinctions and dividing lines as much as she does a vacuum. Another difficulty is the order in which these groups of reflexes should be arranged. The evolution of individuals differs within great limits, and reflexes that are firmly organised in one are incompletely organised in another. The groups that we offer are merely provisional, and are put forward as an indication of the line on which we ought to proceed in building up the reflexes, and more especially as a guide in the examination of cases; for it is by such a regular and orderly examination of the reflexes in connection with sight, hearing, and all the senses, and by endeavouring to distinguish the different associations that are concerned in them, that we may hope to arrive at a satisfactory knowledge of the mental condition of a patient.

There is need of a word of caution here against the error of concluding that a reflex is lost because it is not immediately forthcoming. It may be held in check and prevented by some inhibitory power. What we call the will may from sheer contrariety set itself to counter-work the ordinary reflex

actions, or it may be urged to this course by powerful motives. Again, the attention may be so concentrated on some particular thought, that the visual impressions die away, and lack strength to call forth the usual reflex; the fixed ideas of certain forms of insanity furnish us with the best illustration of this variety, which also includes cases of abstraction, reverie, dominant ideas, &c. Or it may be that the reflexes, especially the higher ones, are not obtained, because of the want of attention. It is important to remember and avoid these sources of fallacy, in deciding on the condition of the reflexes.

We can imagine objectors saying, "Well, suppose you get the so called visual reflexes properly grouped together, and something like a natural classification formed, of what use will it be? You are not dealing with sight alone; in your visual reflexes there is a regular jumble of sight, touch, hearing, the kinæsthetic sense, in fact of all the senses, with a few of the appetites, several distinct instincts, and the whole group of the higher faculties of reason, memory, judgment, attention, &c. —such a jumble, in fact, that it is quite impossible to say where sight begins or where it ends, or in some of the reflexes to see what sight has to do with them at all."

With most of this criticism we should cordially agree. We are not dealing with sight alone, with sight defined as sensitiveness to light, or mere ability to see form and colour. Sight has in these brain questions no such restricted meaning. It is recognised that there is sight and sight, and we speak of intellectual or psychical blindness, word-blindness, and other forms of blindness. We have, in a word, to deal with sight and its associations; with the visual pictures that are called up by the sight of an object as well as with the tactile, auditory, motor (or, as Bastian would say, kinæsthetic), and other memories that are aroused. Such an examination is, unfortunately, fraught with complexity, but complexity is inherent in the problem. The various senses are knit together in the brain in a network of associations, the plan of which we can never hope fully to know; but we shall do better if we work at the fringe of this network, and seek to obtain a knowledge of it rather than weave for ourselves a new fabric, and delude ourselves with the idea that it is nature's product. The method

of the visual reflexes has been selected, not because it furthers any *à priori* notions of well-defined cerebral localisation, but because it provides us with a satisfactory view of one department of a patient's mind. The splitting up of these reflexes into their component parts, and the determining in cases where they are absent, whether the affection is on the motor side, or the sensory, and in the latter case the deciding as to how far the appearances are due to disorder of the purely visual associations, or of the associations with other senses—these are problems of varying difficulty in different cases. Sometimes they are comparatively easy of solution; at other times it is impossible to arrive at a satisfactory conclusion, owing to the condition of the patient, or to our present lack of knowledge. Take, for example, the reflex of reading at sight. Does the sight of a word call up the sense at once (by recalling we will say, the picture of the object), or only after arousing the corresponding auditory sound; or, yet again, only after arousing a suppressed articulation of the word? Until these points are settled, we cannot hope to analyse satisfactorily cases of word-blindness.

Before passing to a consideration of the various groups of reflexes, we would again emphasise the importance in case-taking of being dissatisfied with such general terms as hebetude, stupor, dementia, enfeebled intellect, impaired faculties, &c. We should describe the facts that come before us, as far as possible grouping them around the senses they primarily concern. We would also note the practical importance of investigating the condition of the eyes separately as well as in combination.

We shall in our view of the visual reflexes confine ourselves to what may be called presentative sight as distinguished from re-presentative sight; in other words, we shall occupy ourselves with the visual associations that the sight of an object calls forth, and not with those that are called forth by auditory or tactile impressions, or by the internal rehearsing of a name. We shall further limit our attention chiefly to those conditions implying paralysis or loss of function, and shown by absence of the visual reflex—leaving untouched the consideration of those more complicated conditions characterised by perversion

or exaggeration of function, excluding, therefore, hallucinations, and illusions of sight. The examples we shall give, particularly those in sections 2 to 8, are taken mainly from observations made by Fürstner,[1] Reinhard,[2] Stenger,[3] Zacher,[4] and ourselves. We do not deem it necessary to give more than these general references.

We premise that in the cases examined there is no purely optical defect, no defect of media, of the retina, or of the optic disk.

(1.) *The Light Reflexes.*—Under this head we may conveniently include three phenomena:—the contraction of the pupil when a bright light falls on the retina; the dilatation of the pupil when the eye is removed from a bright to a dull light; and the emotional effects of light. The stimulus may be pleasurable and cause a smile, as is often seen in infants; or it may be too powerful, and be followed by an expression of pain, movements of general uneasiness, closure of the eyelids, aversion of the head, and raising of the hands, to the eyes.

These three phenomena should be investigated separately. The contraction of the pupil to light is the lowest and best organised of the visual reflexes; its relations to the other reflexes of this group and to the higher reflexes are not so clearly made out as is desirable. The pupil may contract to light, and yet there be no emotional manifestation of pain or pleasure, even though a strong light is shone into the eye; and conversely, the latter may be present and the former absent. Another point to be considered is the possibility of a retinal impression stimulating higher centres that inhibit this lower reflex. In studying these reflexes we must remember to exclude the contraction of the pupil that occurs on convergence of the eyes; the pupil may contract well on convergence, but badly to light.

(2.) *Objects fixed and followed by the Eye.*—The characteristic of this group of reflexes is that the eye does not gaze vacantly

[1] Fürstner, 'Archiv f. Psych.' viii. p. 162, and ix. p. 90.
[2] Reinhard, 'Archiv f. Psych.' ix. p. 161.
[3] Stenger, 'Archiv f. Psych.' xiii. p. 218.
[4] Zacher, 'Archiv. f. Psych.' xiv. p. 489.

into space if an object is held before it or crosses its field of vision, but the object is looked at and its movements followed; it is seen.

The fixing of an object by the eyes is by no means the simple act that it appears. Leconte points this out with great clearness in his work on Sight, and he shows that in every act of looking at an object, there are involved, besides the innate idea of direction, and the principle, almost as innate, of corresponding points, three adjustments,—the focal adjustment, whereby each eye obtains a perfect image of the object; the axial adjustment, whereby the axes of both eyes meet in the object; and thirdly, the pupil-adjustment, seen in its contraction on convergence of the eyes. Now each of these adjustments is based upon a visual reflex, so that we have the retino-ciliary reflex, the retino-ocular reflex (as we may term the arc that connects the retina with the ocular muscles), and the special retino-iridal reflex. Further, these adjustments or reflexes are grouped together and co-ordinated in the act of looking at an object, and with them are associated certain movements of the head and neck. Each of these several reflexes may be disturbed, and also the group of co-ordinated reflexes; but these are affections that belong to the domain of the ophthalmic surgeon, and we shall do no more than mention them. Looking at things and following them with the eyes gives rise to ideas of form, size, and distance; indeed we may say that these ideas are present potentially or actually in every visual picture. Are there cases in which these fundamental ideas are seriously affected? It is only in a few exceptional cases that we are able to arrive at a conclusion. Infants who have not commenced to check their visual by their tactile experiences have the vaguest ideas of distance, and Cheselden's celebrated patient saw things flat as in a picture, and said that all objects seemed to touch his eyes. We are not aware of any observations on the adult which indicate an affection of these fundamental ideas.

Let us now consider the cases in which this power of seeing objects is impaired or lost. We need scarcely say that the retina is a surface, not a point, and that consequently sight may be lost in one part of the retina, and retained in another.

This leads us to affections of the field of vision, and of these hemianopsia is the most important.

We can only draw attention to a few of the main points in connection with *hemianopsia*. We should in every case determine, by the perimeter if possible, whether vision is still retained for some distance all round the fixation point, or whether the line of demarcation between the two divisions of the field of vision passes through this point. We should further determine the degree of blindness in the affected portion of the retina, ascertaining whether there is absolute amaurosis, or whether a feeble luminous sensation can still be aroused by a strong light; or, if the patient is unable to give this information, whether a reflex can be evoked by the approach of a lighted taper or by other means. We should also inquire whether the affected field of vision appears to the patient like a dark or grey surface, or whether it is an absolute blank of which he is as unconscious as of the existence of the blind spot. Investigation of these points is likely to throw light on the localisation of the lesion that produces hemianopsia. For example, we sometimes have hemianopsia accompanying the apoplectic or convulsive seizures of general paralysis, and as the lesion in these cases is regarded as cerebral, we should expect an amblyopic rather than an amaurotic condition of the affected field of vision. And yet in several of these cases that have come under our notice there was absolute blindness in the affected part of the retina; no objects were seen, a lighted match thrust up the eye had no effect. Wilbrand cites some interesting cases of recovery from hemianopsia with gradual appearance of the visual reflexes, but for fuller information on this subject we must refer the reader to his work.[1] Before dismissing this subject of the visual field, we may add that partial hemianopsia or sector-shaped defects of the field should be looked for, and also central and peripheral defects.

Another visual affection in which the power of fixing and following objects is lost, is *amblyopia*. Amblyopia, or dulness of vision, is a somewhat vague and unsatisfactory term. Its essential feature is diminution of the acuity of vision, with absence of such ocular defect as would account for it. In

[1] Wilbrand, 'Hemianopsie.' Berlin, 1881.

amblyopia, objects may have to be brought up to the eye to be seen, small objects may not be seen at all, colours may be misty and indistinguishable, and objects appear pale and colourless. It is evident that in such a state a patient will be very apt to make mistakes; he may easily mistake a sovereign for a shilling, and may fail to recognise many objects that are placed before him. It is then of fundamental importance in the method of examination we are pursuing that this question of the existence of amblyopia should be settled in every case, for the absence of the higher reflexes that we shall soon consider may depend on the simple fact that the objects are not distinctly seen, and not be due to any intellectual defect or defect of associations. A word of caution is necessary as to cases of unilateral amblyopia. Wilbrand is sceptical of the existence of such cases in cerebral affections, although they have been described, and he dwells on the importance in cases of this sort (*a*) of a careful perimetric examination, as hemianopsia is so easily overlooked, and (*b*) of an enquiry into the previous visual acuity of the patient, as amblyopia may have been present before, enjoining us to remember the combinations of hemianopsias, &c., that may be produced by bilateral brain affections. Amblyopia is one of the results of such combinations.

A third visual affection in which objects cannot be fixed and followed is *amaurosis*. Temporary amaurosis is a condition that deserves further enquiry. Snell[1] records a case of amaurosis fugax lasting two days; and Panas[2] a case of complete bilateral blindness lasting six hours. In the cerebral hemianæsthesia of Türck there is sometimes a temporary amaurosis; and in cases described by Fürstner there was unilateral amaurosis.

(3.) *Objects seen and handled.*—In this group are included the reflexes that connect vision with the simpler movements of the hand. An object is held before the eyes, and it is grasped at; if placed on a table, it is seen and picked up; if the hand is offered, it is seized. For the simplest reflex of this kind there is required, (*a*) sight of the object, as in the previous section,

[1] See 'Ophthalmic Review,' vol. i. p. 399.
[2] 'Gazette des Hôpitaux,' 1881.

(*b*) an idea of its position and distance, and (*c*) an idea of moving the muscles of the arm so as to reach the object. There may be no idea of what the object is; anything would be handled.

Loss of this reflex would be shown by the fact, that though objects are seen and followed with the eyes, they are not grasped by the hands. Before we can conclude such loss, however, we must remember the cautions already given, as to the state of the attention, presence of stupor, etc. As instances of disorder of this reflex, we may mention those cases in which grown-up idiots grasp at objects beyond their reach; a patient may grasp first at one side of an object then at the other, before succeeding in the attempt to seize it. Carpenter[1] cites an interesting case of a child three years old operated on for congenital cataract; who clearly recognised direction, but had little idea of distance; when told to lay hold of a watch, he groped at it like an infant lying in its cradle. These disorders are distinct from the clumsy grasping movements that depend on impairment of the kinæsthetic sense.

(4.) *Objects associated with pain recognised and avoided.*—This group implies an advance on the foregoing, in that certain sights are associated with painful experiences, and the movements are regulated by the memory of these experiences. Thus, in grasping objects, it is discovered that fire burns, an object thrust up to the eye is found sometimes to hit it; a sharp object is felt to cut. And in future an object suddenly approaching the eye causes closure of the lids, aversion of the head, protective movements of the hands; a lighted match is no longer seized; the sharp edge of a knife is shunned.

If the sight of these objects does not recall the associated idea of pain, we find an absence of these movements of avoidance. It is easy to quote instances of this. A red-hot poker may be brought up to the eye, and the patient gazes at it quite easy-minded and unapprehensive; he may even attempt to take hold of it. If he sees a lighted match blown out, he may attempt to put the glowing end into his mouth. Pretended blows cause no blinking.

(5.) *Food recognised and eaten.*—In this group we have a still

[1] 'Mental Physiology,' p. 181.

further advance. Among objects that are seen, some are recognised as good for eating, and they are seized and eaten—by the hands in the more primitive condition, with the aid of knife and fork, etc., in the more civilised. The sight of the object recalls the old associations formed by eating it, the associations of its taste, touch, etc. If these associations are lost, various curious phenomena present themselves. A person may not recognise food by sight, and never take it so long as it is simply held before him, being in fact in a condition no better than Flourens' pigeons, which would have died of hunger with food before them; but he will recognise it immediately it touches his lips, and devour it with delight. Or he may forget, and conceive some things to be food that are not; he may bite a watch, a flower, a key; he may be unable to distinguish soap from bread till he has tasted it; he may eat paper and drink ink; some bite everything held before them.

Some idiots and dements are not able to feed themselves, though they follow the spoon with interested eyes. Other patients are only able to feed themselves with their fingers, the sight of a knife and fork not suggesting the way to use them, or they may bungle in the use of them; they may eat soup with a fork, or try to cut with the back of their knife, or drink from their bread, and raise the saucer to their lips and bite it.

We have been considering only the commonest kinds of food and instruments of eating, such as are in daily use. If the associations are less organised, if, for example, a glass of acid were swallowed for a glass of water, or a poison-berry were taken in mistake for a hip, the act falls into a higher category.

(6.) *The Visual Reflexes in walking and the allied movements.*—In a healthy adult sight contributes little to those guiding sensations which play such an all-important part in maintaining the balance of the body; the sensations of sight are only seriously called on when the kinæsthetic sense is impaired. Sight, however, is in constant requisition in locomotion. It enables us to walk in a straight line, avoid obstacles, go up steps readily, sit down safely, etc. When the visual associations that enter into these movements are absent or impaired,

we find the patient stumbling against obstacles, jostling against people, running against lamp-posts, doors, or walls. In one of Reinhard's cases the patient ran against a door and stumbled against the furniture in her room, sometimes even though it was staring her in the face. She seemed incapable of judging distance, and did not know when she came up to an object, often the first intimation of the fact was her knocking against it. When she did notice an obstacle she gave it an absurdly wide offing.

Obstacles are seen, but they no longer suggest the movements that are required to avoid them. Past experience, in other words old associations, are not available, or, at all events, are not made use of. The more unusual the obstacle, the less likely is the patient to avoid it. Though able to walk about with a degree of safety at home, he may be quite unable to steer his way in an unknown house, or on uneven ground. Sight of a train noiselessly approaching, or of an open grating in a street, may suggest no danger, and cause no alteration in his course. The impairment of this reflex is also seen in a person's attempting to get on a chair, or jump down from one. There seems a want of association between the judgment of the distance involved and the movements to attain this distance.

(7.) *Objects recognised and used properly.*—In this group the test of recognition is the ability to use properly. In applying the test it is important to eliminate the complications that arise from the association of sight with the other senses, especially with touch. For example, a key is held before a patient; if he points to a door and makes a turning movement with his hand, it is clear that the mere sight of it has suggested its use. But if the key has to be put into his hand, and he has to examine it with his fingers before he knows what it is, then we have to deal not with sight alone, but with sight associated with touch. A good example of this is seen in a case of Critchett's, cited by Carpenter:[1] A young woman, blind from birth, whose sight had been imparted by an operation, was not able to ascertain what an object was by sight alone; she would describe the shape, colour, glistening appearance of

[1] Loc. cit.

a pair of scissors, but could not tell what they were till she had put her fingers in them. Here the sensations of touch contributed more than those of sight in determining the use of an object.

The sight of an object calls up ideas of weight, touch, smell, taste, sound, and it is by these associations that an object is recognised, and its proper use rendered possible. When these associations are destroyed, the power of recognition and use is lost, and we have one of the conditions to which Zussmaul applies the term "apraxia." Instances of this condition have already been referred to under former heads; we shall now give others that occur in connection with objects a degree less simple.

Objects of everyday use, such as a watch, penknife, book, pipe, thimble, umbrella, etc., are looked at vacantly, and no signs are made that betoken recognition. Even if placed in the patient's hand they are handled strangely, or played with in an idiotic manner; the watch is not put up to the ear, or the knife opened, or the pipe put into the mouth. A match-box with matches in it is turned round and round with an air of helpless ignorance. In one case a patient knew what his bed, a piece of bread, and a fire were, but not what a needle, a shoe, or his clothes were. He may have forgotten the simplest properties of things, and may busy himself trying to bend a piece of wood or iron; indeed, a person in this condition would no doubt, if opportunity offered, good-naturedly stroke a tiger, or attempt to coax a lion. An object may be recognised as a member of a class, but not as an individual. In the higher instances of individual recognition, call is made on finer and more numerous associations, and the act is removed from the group of comparatively simple reflexes at present under consideration. Our knowledge of an object is very much a matter of the associations it arouses.

The recognition of well-known faces, though scarcely falling within the literal heading of this group, may be conveniently treated of here. The patient may have no idea that the people who wait on him are his wife and children; an old friend is not greeted in the accustomed way; a stranger is taken for an old acquaintance. Ribot mentions a case where the physician

was not recognised, though he came every day for fourteen months; and a patient recently told me that his wife and child, who had been visiting him, were good imitations, and well made up, but he was certain they were nothing more—for one thing, the child was not large enough.

It may be that objects are still recognised, but only dimly and with uncertainty; they appear new and strange. For example, a patient's hands may look odd and queer to him, quite different from what they used to look; and familiar places and buildings may seem so strange, that he will doubt whether he has seen them before.

(8.) *The Visual Reflexes in Dressing, etc.*—Dressing is one of our acquired semi-automatic acts, and it requires that particular articles should be recognised, and their use and the order in which they should be put on known. Sometimes we find that the articles of dress are not recognised by sight; a shoe, a hat, a shawl, may be staring a person in the face, and yet suggest no ideas as to their use. He may have forgotten how to put his clothes on; he may spend an hour fumbling at his trousers, and at the end be no nearer the goal than when he started; he may put his waistcoat over his coat, or look aggrieved when a fourth coat in which he is just preparing to robe himself is taken from him. With his eyes wide open he may persist in trying to thrust things into a supposed pocket, though a moment's glance should convince him that there is no pocket there. He may, like a celebrated Scotch professor, go about with his wife's white stocking on one leg, and his own black one on the other, and be continually doing similar acts. He may lie down on a chest of drawers, and think it is a sofa.

(9.) *The Visual Reflexes in Handicrafts and Arts.*—The reflexes embraced in this section are on a distinctly higher level than those previously considered, and it is correspondingly difficult to distinguish the part that sight bears in them. Still, sight and its associations enter largely into such actions as sewing, drawing, reading music at sight, boxing, digging, and the rudimentary operations of most manual employments; and it is of importance to test a patient in respect to actions of this kind to which he has been accustomed. If he is unable to

perform them, bungling simple work, and failing in what used to cost him scarcely an effort, we have reason to conclude, due regard being paid to other disturbing influences, that the visual reflexes are impaired, and we have a valuable indication as to the state of the patient's brain. As an example of disturbance under this head, we may mention the case of a man who used to sketch well, but who after a cerebral attack would draw a man's head that a child might be ashamed of. Other examples will readily suggest themselves to the reader.

(10.) *Gestures understood.*—Pantomime is the simplest form of language, and is almost universal in its application; it falls therefore to be considered before the higher reflexes that are concerned in naming objects and in reading. We should ascertain whether the patient acts properly when a hand is held out to him. Does he protrude his tongue when we point to his mouth, or go so far as show him our own? Does he come to us if we beckon him, or does he look in the direction we point? Will he produce his knife when we go through the pantomime of sharpening a pencil, and point to his pocket? It is unnecessary for us to say more on this head, the absence or impairment of the power of understanding simple gestures betrays affection of certain visual reflexes.

(11.) *Objects named.*—From our earliest childhood we are accustomed to associate the sight of many familiar persons and things with their names. When this association fails, and the sight of the object does not recall its name, it is only another way of expressing the same fact to say that a visual reflex is in fault. In such a case, we have to inquire what class of objects cannot be named; whether those that we only rarely see and name, or those that we see and name almost every day of our lives; thus a man may have forgotten the names, bread, water, grass, or may not remember his wife's name. In other cases the power of naming objects may not be wholly lost; the patient may give some name closely allied, or may be able to find the name of the genus though not of the species, or may enter into a description of the article and seem to know everything about it but its name. Again, sight alone may fail to suggest the name, but sight, aided by some other sense as touch, hearing, etc., may recall it. The subdivisions of this

group and the different points to observe are too well known to need further notice here.

(12.) *Objects counted and figures read.*—It should be seen whether the patient can tell or indicate the number of fingers shown him, or can count correctly a series of objects laid out before him. We may find that the patient is able to count a number of large objects, but is unable to count small objects that are placed close together; for example, a string of beads. The presence of amblyopia would explain this; but there seem to be cases where the individual objects are distinctly enough seen and yet a series cannot be followed, owing either to some fault of nervous conduction, or to some central defect, probably a kind of irradiation.

We should also determine whether the patient is able to read figures. Can he tell individual figures, and combinations of them? 2758, for example, may be read 2700, 50, 80; and there are many varieties into which we need not enter. Can he tell the time on a watch? Can he do simple addition? Mlle. Skwortzoff[1] cites a case in which the power of reading figures was retained, while that of reading letters was lost.

It should also be noticed whether the value of the numbers is appreciated, or whether it is merely the names that are remembered. An estimate of this can be formed by the appearance the individual makes in a game at dominoes or cards.

(13.) *The Visual Reflexes in Reading.*—A person may see a page of print, know that it is print, be able to tell when it is upside down, notice the paragraphs, italics, capitals, and even be able to find a given place again, and yet be quite unable to read; just as he may recognise handwriting, or know his own name without being able to decipher a letter. This is the affection known as word-blindness.

A step higher, and he is able to name individual letters, or syllables, or words. He may be able to read some words and not others. He may mispronounce letters and words, or give altogether wrong names to them. He may skip words in reading, or pass over lines, or go from the middle of one line to the next. He may read in this way, and have scarcely an idea of the meaning of what he reads.

[1] Mlle. Skwortzoff, 'De la Cécité et de la Surdité des Mots.' Paris, 1881.

Still a step higher, and he can not only read, but can understand what he reads. Individuals who have lost this power remind us, says Mlle. Skwortzoff, of persons who remember the rules of pronunciation of a foreign language, and are able to read it without understanding a word of it. Short simple sentences may be comprehended, while complicated ones only bewilder. If the patient is aphasic, we can test his ability to understand what he reads by writing down some simple request and observing whether he complies with it. It is not sufficient to get him to turn over the page while we are reading, or point out the passage read, for we must remember that a patient may recognise a word when it is uttered and yet be unable to read it without this help.

(14.) *The Visual Reflexes in Writing.*—We must distinguish between ability to copy writing, and ability to write spontaneously or from dictation. Copying is in some cases merely an instance of drawing; indeed the patient may have so forgotten the once almost automatic art of writing, that he may begin at the last letter of a word instead of the first. We should examine every patient and see whether he is able to copy written or printed text, and can convert the latter into written characters.

Writing spontaneously or from dictation is a complicated process which it is not easy to analyse satisfactorily. Many educated people can write with their eyes closed, and many cases have been reported of patients being unable to read what they had just written. As we are now dealing with sight only so far as it is the starting-point of a reflex, we must limit ourselves to the consideration of the influence sight exercises on the act of writing. Under the influence of sight the letters are kept at proper distances from one another, an approximation to a straight line is maintained, and glaring errors of orthography or composition are detected. In some brain-diseases, the reverse of all this is observed; for example, the letters are badly formed, or heaped one on the top of the other, or the lines may not be kept to, or a commencement is made in the absurdest places, or striking errors pass unnoticed. Examination of the writing of a patient should not be omitted where an inquiry is being made into the condition of sight.

(15.) *Higher Visual Reflexes.*—We are now arrived at the more complex and less organised reflexes which it is difficult to group together satisfactorily, and we shall be content with the easier plan of pointing out the steep and thorny way of investigation instead of treading it ourselves. Are the more complex and intricate visual presentations understood by the patient as they once were? Can he remember his way about in his own neighbourhood or in less familiar districts? Does he grasp the significance of what he sees happening around him? If, for example, he saw two men fighting desperately, would he recognise the fact, or merely stare at them, or think they were amusing themselves? Does he appreciate changes of facial expression and demeanour? Is he as alive to beauty of form or colouring? Does he understand symbols and symbolic actions? In other words, are the same delicate associations called up by the sight of objects as formerly were? These higher visual reflexes carry us into the highest reaches of the mind, and their analysis and due appreciation naturally present difficulties that are great even where normal brain-action is under consideration. How much greater are they when the problem is overlaid by the vast disturbing influences of disordered brain-action and its offspring, protean Insanity! But are we entitled to conclude that sight is unimpaired until these higher regions of intellectual sight have been explored? For our part we think not, and we would not hesitate to say that if a Wordsworth became as a Peter Bell, no longer feeling the witchery of the soft blue sky, regarding a primrose by a river's brim as but a yellow primrose and nothing more, he would in our opinion be suffering from an undoubted visual defect.

We have confined our attention chiefly to the intellectual side of vision, but in a complete view of the subject regard would also be had to the manifestations of the emotions that are called forth by sight.

To be continued.

AN ANALYSIS OF THE NERVE-PHENOMENA IN A CASE OF ANÆSTHETIC LEPROSY.

BY W. ALLEN STURGE, M.D.

Mr. D. aged 53, consulted Mr. Hutchinson in July 1881, through whose kindness I was enabled to see the patient. The patient said that as a young man he had good health until the age of 23, when he went to reside in Bermuda. There he had a severe illness, which he believes was a malarial fever of some kind. Shortly after his recovery from this illness he began to suffer from a skin-disease, which appeared first on the front of the abdomen, and gradually spread to the back, arms and legs; but, so far as he can recollect, did not affect the hands and feet. This eruption lasted for many years, and then gradually subsided. At the present time there are a few patches on the back, and on the right arm, which are not distinguishable from ordinary psoriasis. The patient states, however, that he was seen by Dr. Brown-Séquard when the eruption was at its height, and that he pronounced it to be the eruption of leprosy.

Not long after the onset of the eruption he began to suffer from hyperæsthesia of the extremities. The feet became so tended that it inconvenienced him to stand upon them, and it hurt him to clench things tightly in the hands. The hyperæsthesia gradually extended up the limbs. As time went on, this hyperæsthesia became less and less marked, until at last it was replaced by numbness, at first slight, but gradually increasing until it became complete. The onset of anæsthesia was accompanied by the complete cessation of the sweat function in the parts of skin affected by it.

As the modification in sensation has progressed, he has gradually noticed that his limbs were getting weak, and that some of the muscles were wasting.

At the present time there is complete anæsthesia over the whole extent of the upper and lower limbs, and over areas of the trunk adjacent to the limbs. The greater portion of the trunk is, however, free from it. Between an anæsthetic part of the skin and the adjacent healthy portions there is a border of skin, from half-an-inch to an inch wide, where there is a moderate degree of hyperæsthesia, and these borders obviously point to the fact that the disease is still slowly advancing. Wherever the skin is anæsthetic it is thin, dry, more or less shiny, and the sweat-glands and hair-follicles appear to be completely atrophied.

There is complete atrophy of all the small muscles of both hands, which have assumed the most marked "main-en-griffe" position. In the left arm there is complete wasting of all the muscles on the back of the forearm, including the supinator longus; and there is a slight degree of wasting in the muscles of the front of the forearm. The biceps, brachialis anticus and triceps are nearly or quite healthy, but the deltoid is markedly wasted. In the right arm there is no wasting of the muscles on the back of the forearm; a slight degree of wasting of the muscles on the front of the forearm; none of the biceps, brachialis anticus, or triceps; but marked wasting of the deltoid. The other muscles about the shoulder-joint and the serratus magnus were healthy on both sides. There was wasting of the small muscles of the feet; complete wasting of the muscles on the front of both legs; but little, if any, of those of the calves or of the thighs.

The muscles of the back, chest and abdomen were healthy. The face presented a curious condition; there was complete anæsthesia of the forehead and eyelids, but the rest of the face and the scalp were ordinarily sensitive to touch. There was a corresponding atrophy of all the muscles beneath the anæsthetic skin, viz. the occipito-frontalis, the corrugatores supercilii and the orbiculares palpebrarum. Some years ago he began to experience difficulty in shutting his eyes, which increased, until all power of moving the eyelids had gone. From that time he began to suffer from severe inflammation in the eyes, and at last, after many remedies had been tried and one or two operations had been performed, he lost his

sight altogether, and to prevent irritation the eyelids were sewn together. The muscles of the rest of the face are healthy, as are those of the tongue, throat and larynx, in proof of which he states that his voice is exceptionally strong, and that he has no difficulty in making himself heard clearly at large public meetings.

It is a point of much interest that, although the skin of the limbs is so completely anæsthetic, there is apparently little or no anæsthesia of the deeper structures. He is able to feel *deep* pressure quite easily, and he even feels a pinch quite well, if other structures than the skin be involved. I regret that I did not observe whether this sensitiveness of the deeper structures was also present in those places where the muscles were extensively atrophied. For reasons that I shall presently state, I think it would be important in any similar case to note this.

His brain power throughout has been excellent, and, notwithstanding his crippled condition, he is a distinguished member of one of the learned professions.

The diagnosis in this case was that it was an old-standing case of leprosy in which the anæsthetic symptoms were exceedingly well marked, whilst the skin symptoms were only moderately so; in other words, that it was a case of non-tuberculated anæsthetic leprosy. I shall endeavour to make an analysis of the phenomena present, with a view of throwing further light, if possible, upon the pathology of the nerve-phenomena of the disease. I regret that in the single, somewhat hurried interview that I had with the patient I omitted to note several points of importance for this purpose; but I think we have sufficient data to enable us to arrive at definite conclusions in regard to some of the questions which may arise.

We have to consider, (1) the primary seat of the nerve lesion; (2) the course followed by it in its progressive advance.

The primary seat of the nerve lesion.—The questions which present themselves in this connection are: (1) Is the lesion *central*, i.e. in the brain or spinal cord? or is it *peripheral*, i.e. in the nerves? (2) If peripheral, is it (*a*) in the trunks of the

nerves? (*b*) in the finer nerve branches? or (*c*) in the peripheral terminations of the nerves?

Answers to these questions may be sought for by an examination—(1) of the sensory phenomena, (2) of the motor phenomena.

1. *The sensory phenomena.*—The patient's description of his sensory symptoms was that he first noticed hyperæsthesia of the extremities; the feet were tender, and it was painful to hold things firmly in the hands. The hyperæsthesia gradually extended up the limbs, and after a time it slowly diminished, being replaced by anæsthesia, which ultimately became complete. On examination, we found that all four limbs were completely anæsthetic, together with a strip of skin in the adjacent portions of the trunk. The anæsthetic areas were still bordered by a band of hyperæsthesia.

The first thing to observe about this account is that the mode of invasion of the anæsthesia is wholly unlike anything that is met with in central disease. The patient was quite clear in his statement that it began at the extremities of the limbs, and gradually crept up towards the trunk, and this statement is borne out by the border of hyperæsthesia still existing. It is also quite unlike the history we should get if the nerve-trunks themselves had been primarily involved. In that case we should have had the anæsthetic patches taking shapes corresponding to the distribution of sensory nerves. It may be said that we have only the patient's own statement that the anæsthesia did not come on in this way, and that he would be unlikely to notice the exact mode of onset of his numbness. Against this I would urge that he was a man of high and cultivated intelligence, that the anæsthesia has been gradually advancing for more than twenty years, and that he has carefully watched his own symptoms during that time. The present margin marking the advance of the disease shows no sign whatever of following the lines of certain nerve distributions.

Thus then we are driven to the conclusion, that the primary seat of the nerve lesion is not in the central nervous system, nor in the trunks of the nerves. It must therefore have been in the ultimate branches of the sensory nerves, or in their peripheral terminations.

Another point in favour of the conclusion, that neither the central organs nor the trunks of the nerves were primarily affected, is the fact to which I drew attention, that although the skin was absolutely anæsthetic, yet he was able to feel deep pressure quite easily, and he even felt a pinch quite well if other structures than the skin were involved. Unfortunately I have no note of the relative deep sensibility of parts where the muscles were healthy, as compared with those where they were atrophied. My conviction is that there was no sensation in the atrophied muscles. In anæsthesia from central disease, no such condition as this is observed. A limb is wholly or partially anæsthetic in its entirety; or if, as sometimes happens in locomotor ataxy, a district of a limb becomes anæsthetic, we have no such superficial anæsthesia as that here described.

Similarly in disease of a nerve-trunk, complete or partial anæsthesia of all parts superficial and deep supplied by that nerve ensues; but here also a purely superficial anæsthesia is never met with.

Thus then all the sensory phenomena in this case point to the conclusion, that it is the peripheral terminations of the nerves that were primarily involved.

Before passing on to the next division of the subject, viz. the motor phenomena, let us see whether we can get evidence to prove that the mode of invasion above described is a usual one in cases of non-tuberculated leprosy. For this purpose I cannot do better than refer to the masterly treatise of Dr. Hillis on "Leprosy in British Guiana."

In his chapter on non-tuberculated leprosy, Dr. Hillis describes the following cases:—

(1.) A man, æt. 64, affected for 8 years (Case XXI. p. 84). There is cutaneous anæsthesia of both arms from the fingers to four inches above the elbow-joints, and from the toes to the ankle-joints inclusive.

(2.) A man, æt. 47, affected for 9 years, (Case XXVI. p. 89). He had complete cutaneous anæsthesia up to the insertion of the deltoid in the left arm, and in the right arm up to the elbow-joint in front, and to the middle of the back of the forearm. Complete cutaneous anæsthesia of the feet and legs up to the knees.

(3.) A man affected for 15 years (Case XXXIII. p. 97) is described as having lost sensation in all the limbs as far as the knees and elbows.

Now when we consider how various are the nerves which supply sensation to the skin of the limbs, and from what different sources they are derived, it will be at once evident that no affection of nerve-trunks would give rise to the distribution of anæsthesia described in these cases. I am therefore justified in quoting them as further proof of the conclusion at which I above arrived. I must however state, that Dr. Hillis believed that he could, in certain cases, trace a connection between the seat of cutaneous phenomena and the distribution of nerve-trunks. I gather, however, from his remarks, that he has more especially noticed this in the distribution of the eruption, rather than of anæsthesia, a point upon which the case I am reporting offers at the present time no evidence. Thus he says: "Although in some cases it would appear as if the patch [of eruption] corresponded to the distribution of a nerve, such is not always the case. I have, however, been struck with how frequently the course of the musculo-spiral nerve is apparently taken by this eruption" (p. 74). Of two cases, however, where the eruption was supposed to take this course, I find that the report is too vague to justify us in laying much stress on this point; and when we consider the scattered and serpiginous nature of the patches of eruption, it would not be surprising that some of the patches might appear to follow the cause of some one nerve. But, however it may be with the eruption, in no case that he describes did the anæsthesia correspond to the distribution of one nerve; where it was not simply patchy and thoroughly irregular, it in nearly every instance assumed the form described in the case above reported, and in those quoted from his own book; viz. a gradual creeping up the limbs, without reference to the nerve supply.

I now come to the next branch of the subject, viz.:

2. *The motor phenomena.*—The mode of onset of the muscular atrophy in the limbs differed widely from that of the anæsthesia; for whereas we saw that the latter advanced without reference to the geographical distribution of nerves, the

muscles were atrophied by groups, certain groups being intact, whilst other groups in their near neighbourhood, and placed very similarly as regards distance from the centre and in relation to the anæsthesia, were completely atrophied. This distribution of the atrophy points to the conclusion, that the nerve lesion giving rise to the atrophy was situated either in the spinal cord or in the trunks of the nerves. At first sight, it may not appear to be an easy thing to decide whether a given muscular atrophy is due to a central (spinal cord) lesion, or to disease of the trunks of the nerves. There is, however, one infallible test which will, I believe, be found never to fail, and which is not generally known. I refer to the distribution of the atrophy, which is quite different in the two cases. When the trunk of a nerve is affected in such a way as to produce atrophy, all the muscles supplied by the nerve below the seat of lesion will be equally involved—muscles belonging to different groups, and having little or nothing in common, beyond the fact that they are supplied by the same nerve. In the case of spinal-cord disease, those muscles will tend to be simultaneously affected which are associated together in the performance of certain co-ordinated movements, whether they be supplied by the same nerve or not. This is a statement which can, I believe, be confirmed by a careful analysis of the distribution of the atrophy in any case of muscular atrophy where the distribution is partial. It was clearly proved by two cases, which formed the basis of a paper read by me before the Medical Society of London.[1] In any given case of atrophy of a limb, the muscular grouping according to nerve supply should be carefully thought out and compared with the grouping of the atrophied muscles. In the case of the arm, this is done with great ease, for there is a well-marked group of muscles co-ordinated for a single purpose, viz. to bend the elbow, which does not correspond to the grouping, according to nerve-supply. Three muscles take part in this action—the biceps, the brachialis anticus, and the supinator longus; the two former being supplied by the musculo-cutaneous nerve, and the latter by the musculo-spiral. Disease of the musculo-cutaneous nerve will therefore cause

[1] 'Proceedings of the Medical Society,' vol. v. p. 357.

atrophy of the biceps and brachialis anticus; without the supinator longus; whilst disease of the musculo-spiral will cause atrophy of the supinator longus, without the biceps and brachialis anticus. Now it has been shown, on the other hand, that disease of the spinal cord giving rise to atrophy of any one of these muscles gives rise to atrophy of both the others also; whilst in disease of the spinal cord giving rise to atrophy of the muscles of the back of the forearm (which are supplied by the musculo-spiral nerve), the supinator longus, which is also supplied by that nerve, will not be atrophied unless the biceps and brachialis anticus are also involved.

It fortunately happened that in the left arm of our patient the exact condition was present which is required to enable us to make use of this criterion; for the muscles of the back of the forearm were completely atrophied, whereas the biceps and brachialis anticus were intact.

What was the state of the supinator longus? It was completely atrophied. Hence we may conclude, without fear of error, that the lesion giving rise to the muscular atrophy is situated in the trunks of the nerves and not in the spinal cord.

Thus we are brought to the conclusion, that the affection of the sensory nerves is independent of the distribution of the nerve-trunks, and that the primary lesion giving rise to it is situated in the peripheral terminations of the sensory nerves; whereas the motor affection corresponds with the distribution of the nerve-trunks, and that the lesion giving rise to it is seated in those nerve-trunks. The questions arise:

Is there any connection between these two diversely located lesions? and can any explanation be found for this curious mode of election of the disease?

After carefully considering these questions, I believe that the following is the true explanation of the phenomena. It is evident that the morbid process which gives rise to the leprous condition has a strong tendency, in the non-tuberculated variety, at any rate, to advance by continuity of tissue. Dr. Hillis, in summing up this variety of eruption, says: "At first small, the spots subsequently enlarge, and may by coalescing take over the greater part of the body, . . . they

have a tendency to spread serpiginously and peripherally."[1] It is evident that in this form of the disease it is the skin and nerves which have a special tendency to be attacked; and therefore we might expect to see the same tendency to advance by continuity of tissue in the case of the nerves, as in the case of the skin. I have given reasons for believing that the nervous affection begins at the peripheral extremities of the cutaneous nerves. From this point the disease will creep up the nerve-fibres, and from individual nerve-fibres it will extend upwards to the point where several fibres are collected together into a small nerve-trunk, and up this to the larger branches, and eventually to the main nerve-trunk. The nerve disease is proved by microscopical examination to consist mainly of a cellular new formation, accompanied by some hypertrophy of the connective tissue binding the fibres together. The result of this is that those fibres of the nerve which have not already been injured by the new growth become squeezed, and ultimately destroyed. The branches given off by a nerve-trunk to the muscles supplied by it are, for the most part, independent of the cutaneous branches of the same nerve. As the disease begins in the peripheral extremities of these latter nerves, the motor nerves will not be affected until it has passed up the cutaneous branches to the main branch or main trunk from which the motor nerves are given off. When this has happened, a whole group of muscles will be affected simultaneously—a group deriving its nerve-supply from a common origin.

Let us see whether the distribution of the muscular symptoms in Mr. D.'s case offers any support to this view. To do this, it will be necessary to recall the relations which are borne to one another by the sensory and motor branches of the various nerves of the limbs. In making this review, there are three points to which attention must be drawn in connection with each nerve.

(1.) The position in the limb occupied by the area of skin supplied by the nerve, *i.e.* whether nearer the trunk, or farther from the trunk. Bearing in mind the mode of invasion from the peripheral extremity of the limb towards the centre, it is

[1] Loc. cit., p. 101.

evident that areas of skin further from the trunk will have become affected sooner than those nearer the trunk, and hence that, if my explanation be the correct one, the disease will have had a longer time to travel up the nerves ascending from the areas more distant from the trunk than up those passing from the less distant areas.

(2.) The distance from the peripheral extremity of the sensory nerve to the nearest point where it meets with motor nerves. This is obviously a point of great importance, for if a sensory nerve which may have become affected very early in the disease was given off from the nerve-trunk high up in the limb, as is the case, for instance, with the internal saphenous nerve, the distance to be travelled by the disease would be so great that it might take very much longer to reach the trunk of the nerve, or the motor branches, than in the case of a nerve affected much later in the disease, but offering relatively a much shorter course before reaching motor fibres.

(3.) The size of the sensory nerve both absolutely and relatively to the nerve-trunk or branch from which it springs. A large or widely branching sensory nerve, such, for instance, as the circumflex, might be expected to offer an easier passage for disease than a single small sensory branch of equal length; and the disease would, in all probability, reach the nerve-trunk in a shorter time in the case of the former than in the case of the latter. Whether this be so or not, however, it is certain that disease passing up a sensory nerve of considerable size, and reaching a motor nerve of about the same size, will produce a more rapid and intense effect than if it travels up a sensory nerve of small size, and reaches a nerve-trunk of relatively much greater size. Compare, for instance, disease travelling up the radial nerve to meet the posterior interosseous, and disease passing up the internal cutaneous of the musculo-spiral to meet the main trunk of the musculo-spiral nerve.

Each of these three points must be borne in mind in considering the various nerves. I will begin with

I. *The Nerves of the Arm.*—Here we have five main nerves:

(1.) The musculo-spiral.

(2.) The musculo-cutaneous.

(3.) The median.
(4.) The ulnar.
(5.) The circumflex.

(1.) *The Musculo-spiral.*—The *motor* branches are:

(*a*) Branches to the triceps, given off high up in the course of the nerve.

(*b*) Branches to the supinator longus and extensor carpi radialis longior, given off after passing through the external intermuscular septum, and before the nerve divides into the radial and post. interosseous nerves.

(*c*) The *posterior interosseous* supplying the supinator brevis, the extensor carpi radialis brevior, and all the extensor muscles at the back of the forearm. The posterior interosseous splits off from the radial just below the elbow-joint.

The *sensory* branches are:

(*a*) The *internal cutaneous of the musculo-spiral*, a small nerve which takes its origin high up in the course of the nerve, and passes to supply the skin of the inner and upper part of the arm. Applying the three tests above mentioned, we see that the area of skin supplied by the nerve will be attacked very late in the disease; the disease, however, will not have a great distance to travel up the nerve before reaching the main trunk; and thirdly, that the sensory nerve bears a very small proportion to the main trunk from which it issues.

(*b*) The *two external cutaneous branches.*—These which, taken together, make a moderate-sized nerve, have their origin just after the musculo-spiral has pierced the external intermuscular septum, and before it has given its branches to the supinator longus and ext. carpi rad. longior. They are distributed, the upper to the skin of the lower half of the upper arm in its anterior aspect; and the lower to the skin above and in front of the elbow-joint, and to the outer part of the upper half of the forearm. The

area supplied by them will therefore be affected about the middle period of the disease; the disease will have, on the average, a moderately short distance to travel to reach the trunk of the nerve; and the proportion borne jointly by the nerves to the main trunk, though not approaching equality, is a considerable one.

(*e*) The *radial nerve*.—This is a nerve of considerable size, taking its origin at the bifurcation of the musculo-spiral nerve just below the elbow-joint, and running a long course to supply the skin of a portion of the hand. The area of skin supplied by it would therefore be one of the first to be attacked; but the disease would have a considerable distance to travel before meeting with a motor branch; the proportion borne by the sensory nerve to the motor branches from which it split off is, roughly speaking, one of equality.

If then my explanation of the course of the disease be the true one, how would the muscles supplied by the musculo-spiral nerve be likely to be affected? None of the sensory branches are so placed as to make muscular involvement particularly easy, and there is no nerve which is both affected early in the disease and which offers a short course. The radial, however, is affected early and completely; it is a relatively large nerve, and offers a considerable pathway for the disease, which will therefore be likely, after an interval of some length, to reach the motor branches, and thus to cause atrophy of the muscles at the back of the forearm. The external cutaneous nerves will be affected much later, but offer a much shorter course to the disease, which, roughly speaking, might be expected to reach the trunk of the nerve at about the same time as by the radial, and thus to cause atrophy of the supinator longus and ext. carpi radialis longior. The internal cutaneous of the musculo-spiral will however be affected so late in the disease, and it is so small in relation to the main trunk from which it comes, that it is highly unlikely that any atrophy of the triceps would take place.

(2.) *The Musculo-cutaneous nerve.*—The motor branches of this nerve come off in the upper arm—for the most part in its upper half—to supply the biceps, the coraco-brachialis and the brachialis anticus.

The sensory branches supply the skin of the outer part of the forearm, chiefly in its lower half. The area of skin supplied by it will hence be affected some considerable time after the first onset of the malady, and the distance to be travelled by the disease is very great, the muscular branches taking their rise from a part of the nerve that is far removed from the peripheral cutaneous distribution. Hence it is highly unlikely that any atrophy of the biceps coraco-brachialis and brachialis anticus would be met with.

(3.) *The Median.*—The *motor* branches are:

(*a*) Branches given off just below the elbow to the flexor sublimis digitorum, part of the flexor profundus, the pronator radii teres, the flexor longus pollicis, the palmaris longus, and the pronator quadratus.

(*b*) Branches given off below the wrist to some of the small muscles of the hand.

The *sensory* branches are confined to the supply of part of the hand.

Thus then the area of skin supplied by it will be affected at the very onset of the disease; the nerve up which the disease will travel is one of considerable size; the motor nerves to the small muscles of the hand will be quickly met with, whilst those to the group of muscles on the front of the forearm will be reached very much later. We may therefore expect that the small muscles of the hand will be quickly affected. As regards the others, we may observe that the relation borne by the motor branches to the sensory trunk of the nerve is strictly comparable to that borne by the radial nerve to the posterior interosseous, the areas of skin supplied by the two sensory nerves being situated in precisely analogous positions as regards the first onset of disease; and the branching off of the motor nerves being at almost precisely the same distance up the limb in the one case as in the other. Thus we may conclude, that the chances in favour of the posterior group of muscles being affected are practically equal to those

of affection of the front group of muscles. In a given number of similar cases of disease we might therefore expect that sometimes one group of muscles, and sometimes the other, would be first to be affected, a slight variation in priority of attack in the back or the front of the hand being sufficient to produce this variation in the subsequent motor affection. There is this further remark to be made, that whereas in the case of the median, disease of the upper part of the nerve can only have advanced from the hand, in the case of the musculo-spiral the corresponding advance of disease from the hand will be augmented by disease (beginning later, but having less distance to travel) advancing up the two external cutaneous nerves. Ceteris paribus, therefore, we should expect a more intense degree of motor affection in the case of the musculo-spiral than of the median.

(4.) *The Ulnar nerve.*—The *motor* branches are:

(*a*) Branches to the flexor carpi ulnaris, and half the flexor profundus digitorum. These arise from the trunk of the nerve just below the elbow.

(*b*) Branches coming off below the wrist to supply certain of the small muscles of the hand.

The *sensory* branches are:

(*a*) Branches rising from the nerve in its course down the forearm, to supply the skin of the inner side of the lower half of the forearm.

(*b*) Branches to the skin of portions of the hand.

What we have to say here is very much a repetition of what was said of the median nerve. The nerves to the skin of the hand will be affected at, or soon after, the onset of the disease, which having but a short distance to traverse before meeting with the motor nerves to the small muscles of the hand, will quickly produce atrophy of these muscles. From this point it would take about the same time to reach the nerves of the large muscles of the forearm, as in the case of the median and radial nerves; only that, in the case of the ulnar, the morbid action will be reinforced by the disease passing up from the sensory nerves above mentioned as being distributed to the forearm, which, however, will naturally have been affected

much later than those of the hand. On the whole, therefore, we might expect the upper motor branches of the ulnar to be affected a little sooner than those of the median or the posterior interosseous nerve.

(5.) The *Circumflex nerve.*—Its motor branches go to the deltoid and the teres minor muscles.

The sensory branches supply the skin over the deltoid muscle. With regard to this nerve, we may remark that the motor and sensory portions are, roughly speaking, equal in size, and are intimately related to one another in their distribution, which has not been the case, to anything like the same extent, in the other nerves that have been described. The sensory part of the nerve is short and widely branched. Disease once established in the peripheral termination of the branches, has many roads open towards the centre, and it has but a short distance to traverse before reaching the motor branches. On the other hand, the area of skin supplied by the nerve will have been one of the last to be attacked; but, when once attacked, no long time would, for the above reasons, be required to show motor involvement, which would be likely rapidly to become complete.

Treating the arm as a whole, then, we might expect that all the small muscles of the hand would be early and completely affected. The groups of muscles on the front and back of the forearm, supplied by the median and posterior interosseous nerves respectively, might or might not be involved, the chances in favour of affection of the front group being about equal to those of affection of the group at the back; but if both be affected, the muscles of the back group are likely to be more extensively atrophied than those of the front group. Moreover, if any one muscle of either of these groups be affected, all the other muscles of the same group will also be affected and probably in the same degree. For reasons above given, the chances in favour of affection of the muscles supplied by the ulnar nerve will probably be rather greater than in the case of the two groups of muscles just mentioned. The supinator longus and ext. carpi rad. longior would tend to be attacked by extension of the disease either up the main trunk of the musculo-spiral, or up the ext. cutaneous nerves, the

chances of such attack being roughly equal to, perhaps somewhat less than in, the case of the median and posterior interosseous groups. The triceps would almost certainly escape for the reasons above detailed, and the same remark will apply to the biceps, coraco-brachialis and brachialis anticus. If the latter group were attacked, the brachialis anticus might be expected to be the first to succumb. The deltoid, notwithstanding that the skin supplied by the circumflex nerve would be some of the last to be attacked, would probably rapidly become affected when once the disease had extended up to the height of the shoulder-joint.

In approaching the subject thus from the *à priori* standpoint, I do not think I have said more than the conditions postulated justify. Let us compare the picture thus drawn with that presented by the account—unfortunately only partially complete—of our patient. In him all the small muscles of both hands were completely atrophied. In the left arm the muscles of the group supplied by the posterior interosseous nerve were much atrophied, those supplied by the median being slightly atrophied. In the right arm the muscles supplied by the median were slightly atrophied, whilst those supplied by the posterior interosseous were comparatively healthy.

This distribution of atrophy in the two forearms strictly bears out what was said about the equality of the chances of these two groups of muscles becoming affected. As regards the supinator longus and extensor carpi radialis long., these were completely atrophied in the left arm. Unfortunately I have no definite note of their state in the right arm; and the same remark, I regret, applies to the muscles supplied by the ulnar nerve. I believe, however, that these were completely atrophied in the left arm, but I am unable to say anything of their condition in the right. The triceps was healthy on both sides, as were also the biceps, brachialis anticus, and coraco-brachialis; whereas, on the contrary, the deltoid was markedly atrophied on both sides.

I think that, notwithstanding the absence of a complete definite record of every muscle, which under the circumstances it was not in my power to make, the close correspondence

between the probable muscular affection as worked out *à priori*, and the actual affection as observed, is very remarkable, and goes far to show that the mode of advance of the disease is that which I sketched out in the earlier part of my paper.

II. In order to obtain additional evidence upon the point, I must, at the risk of appearing tedious, apply the same analysis to the nerves and muscles of the leg.

The nerves which we shall have to consider are:

(1) The anterior crural.
(2.) The trunk of the great sciatic.
(3.) The internal popliteal.
(4.) The external popliteal.

(1.) *The anterior Crural.*—The *motor* branches supply the powerful muscles on the front of the thigh, and are very large.

The *sensory* branches are:

(*a*) The middle cutaneous, which comes off high up in the thigh, and supplies the skin on the front of the thigh. The area supplied by it would therefore be attacked late, and disease would have a considerable distance to travel to reach a point where it might affect motor branches.

(*b*) The internal cutaneous coming off high up in the thigh, and supplying the skin of the inner side of the lower third of the thigh and the inner side of the leg. The area supplied by it would be affected earlier than in the case of the last nerve, but disease would have a very long distance to travel before reaching motor branches.

(*c*) The internal saphenous nerve also comes off high up in the thigh, and supplies the skin of the inner side of the lower half of the leg, and some parts of the foot. The area supplied by it would be early affected, but the distance to be travelled by disease would be very great before motor nerves would be affected.

From this we should expect that the motor part of the anterior crural nerve would long remain untouched, the distances to be travelled by the disease being very great

except in the case of the middle cutaneous nerve. This nerve, however, being affected late, and being still a considerable distance from its point of origin, disease would be little likely to reach the trunk of the anterior crural through it.

(2.) *The trunk of the great Sciatic.*—The motor branches supply the biceps semitendinosus semimembranosus, and partly the adductor magnus. The branches are given off, for the most part, in the upper half of the thigh. There are no sensory branches given off by the trunk of the nerve. We should therefore expect that, in consequence of the great distance to be travelled by the disease before reaching the above motor branches, the muscles supplied by them would remain intact.

(3.) *The internal Popliteal nerve and its prolongations.*—The *motor* branches of this nerve are:

(*a*) Branches given off high up in the neighbourhood of the knee to the gastrocnemius soleus popliteus and plantaris.

(*b*) Branches given off to the upper third of the leg to the tibialis posticus, flexor longus digitorum and flexor longus pollicis.

(*c*) Branches in the foot to some of the small muscles of the foot.

The *sensory* branches are:

(*a*) The external saphenous nerve arising a little below the knee, and passing down to supply the skin of the lower half of the back and outer side of the leg. The area of skin supplied by it would therefore be affected comparatively early in the disease, but the disease would have a long course to traverse before meeting with motor branches. The nerve also is very small, in comparison with the main trunk from which it comes.

(*b*) The plantar cutaneus and other cutaneous branches to the foot coming off low down in the course of the nerve. The areas of skin supplied by them would therefore be some of the first to be affected, and the disease would have but a short distance to travel before reaching the motor nerves to the small muscles of the foot.

Thus then we should expect to find early and complete atrophy of the small muscles of the foot, but probably none of the large muscles of the back of the leg, belonging to either of the first two groups mentioned.

(4.) *The external Popliteal and its prolongation.*—The *motor* branches given off by the nerve are :

(*a*) Nerves to the tibialis anticus, extensor longus digitorum and extensor proprius pollicis, given off by the anterior tibial in passing down the front of the leg.

(*b*) Branches to the peronei muscles, given off by the musculo-cutaneous in its passage down the leg.

(*c*) A branch in the foot to the extensor brevis digitorum.

The *sensory* nerves are :

(*a*) Branches from the trunk of the nerve to the skin of the upper half of the back and outer part of the leg. This area would be affected at about the middle period of the disease, and the disease would have to traverse only a moderate distance before reaching the trunk of the nerve.

(*b*) The main trunk of the musculo-cutaneous, which supplies the lower third of the front of the leg and the major part of the dorsum of the foot and of the toes. The area supplied by it would therefore be affected at the early part of the disease, but a considerable distance would have to be travelled before the muscular branches were affected—a distance considerably shorter in the case of the peronei muscles than of the other muscles of the front of the leg. The relative size of the sensory branches as compared with the motor approaches equality.

(*c*) A small branch of the anterior tibial to the skin of the great and second toes, which would be affected very early in the disease, and up which disease would have only a short distance to travel before reaching the nerve to the extensor brevis digitorum, but a long distance before reaching the nerves to the larger muscles on the front of the leg.

Considering the external popliteal nerve as a whole, then, we should expect to find the extensor brevis digitorum to be atrophied early in the disease. We should also expect to find the peronei muscles atrophied earlier than the other muscles on the front of the leg, and much earlier than those at the back of the leg. As regards the relative periods at which the muscles supplied by the anterior tibial nerve would be affected, as compared with those supplied by the posterior tibial, the following remarks may be made: In the first place, according to Quain, the muscular branches given off by the posterior tibial nerve leave that nerve at a point higher up the leg than those given off by the anterior tibial. Speaking of the former, he says: "The muscular branches emanate from the *upper part of the nerve*, either separately or by a common trunk." Speaking of the latter, he says: "In its course along the leg, the anterior tibial nerve gives slender filaments to the muscles between which it is placed." According to Ellis, the branches to the deep muscles of the back of the leg may even come off from the internal popliteal nerve before it has become the posterior tibial. Thus in, at any rate, a considerable proportion of cases, the distance to be travelled by disease up the posterior tibial nerve will be considerably greater than up the anterior tibial, before motor branches to muscles of the leg will be met with.

Again, the anterior tibial nerve runs in the concavity of the angle formed by the leg and foot, whilst the posterior tibial runs in the convexity. When the general arrangements of the ankle are considered, I think it will be at once evident that the anterior tibial is shorter than the posterior tibial, which will still further increase the chances in favour of priority of attack of the motor branches of the anterior tibial.

We are thus brought to the conclusion, that the group of muscles supplied by the anterior tibial nerve will almost certainly succumb at an earlier period than the group supplied by the posterior tibial; whilst the group supplied by the internal popliteal is very unlikely to become involved, unless at a very late stage of the disease.

Treating the leg as a whole, then, we should expect all the small muscles of the foot to be atrophied. We should expect

the peronei muscles to be next affected; and subsequently the other muscles on the front of the leg. Later still, the deep muscles at the back of the leg, and at a somewhat later period those of the calf. We should not expect to find any of the thigh muscles, either front or back, to be affected.

I regret that my notes are not sufficiently detailed to enable me to speak of the condition of all the muscles individually in the legs of my patient, but I have noted that all the small muscles of both feet were atrophied; all the muscles on the front of both legs were completely atrophied; the muscles at the backs of the legs were relatively healthy; and the thigh muscles were apparently in their normal state of nutrition. I think, therefore, that I am justified in saying that the condition of the muscles of the legs bears out the conclusion arrived at by the examination of those of the arms.

I now pass on to a point of great interest, viz. the condition of the face. Here we saw that there was complete anæsthesia of the forehead and eyelids, the rest of the face and scalp being unaffected as regards sensation. Corresponding to this anæsthesia there was atrophy of all the muscles beneath the affected skin; viz. the occipito-frontalis, the corrugatores supercilii, and the orbiculares palpebrarum; all the other facial muscles being healthy.

Now here it is quite evident that the explanation offered above of the muscular affection in the limbs will not apply, for the sensory and motor nerve-supplies in the face have nothing in common, and hence no motor affections could be produced by disease travelling up the sensory nerves. On the other hand, the peculiar distribution of the atrophy points to an intimate relationship between the sensory and motor affections; and points, moreover, to the conclusion that the atrophy was not due to disease in the trunk of the facial nerves, or even of the trunk of its temporo-facial division, a statement which a reference to the plate of the distribution of the nerve in Quain's 'Anatomy' will at once make evident.

In the face, however, we have to do with a muscular arrangement scarcely met with anywhere else in the body, viz. the muscles lie immediately beneath the skin, without the intervention of fascia or other thick tissue; in fact, they may be

said to be practically united to the skin. Hence both they and the nerves connected with them would be peculiarly liable to become affected by any condition leading to infiltration or destruction of the skin. The muscular atrophy in this case is explained, then, by the direct extension of the disease to the muscles by contiguity, or possibly it may have resulted through the intermediary of their motor nerves, the peripheral termination of which may have been involved in the disease, in common with those of the sensory nerves. Possibly both processes have been at work simultaneously; but, however that may be, I think it may be considered as certain that this is the true explanation of the atrophy of these muscles. It is a point of considerable interest, that in this patient we have muscular atrophy produced in two widely different ways; the one being a direct affection from the skin by contiguity, and the other due to involvement of the trunks of the nerves supplying the muscles, by extension of disease up the sensory nerves from their peripheral terminations to their points of junction with motor nerves.

In conclusion, then, if, as I believe, this case may be taken as the type of an ordinary case of non-tuberculated anæsthetic leprosy, we may from it learn the method by which the nervous system becomes affected in this disease.

Leprosy is defined by Virchow to be a disease in which there is a production of a "new granulation tissue" that is effused into the fibro-cellular structures of the body. With the exception of the heart, pancreas, brain, and spinal cord, this neoplasm may invade the fibro-cellular coats and structures of all the organs. Clinical experience, however, shows that there are in the body two tissues which constitute the principal seats of election of the disease. These are the skin and the nerves. In many cases the skin alone is affected, the implication of the nerves being so slight that it may be left out of account. In some cases the nerves are implicated principally, the skin condition being of subordinate importance. In a large number of cases, however, both tissues are affected, the affection of the two tissues being relatively uniform over the body; or the skin affections may be most prominently marked in one part of the body, and the nerve affection in another.

There is nothing strange or unique in this varying elective affinity of the new growth. A similar variation is constantly seen in connection with new growths. Consider the case of syphilitic new growths, which will perhaps be found to be more nearly allied to the leprous neoplasm than any other form of morbid growth. There is scarcely any tissue in the body which may not be attacked by the syphilitic neoplasm; and yet how protean are the ways in which in given cases syphilis attacks the tissues, seizing greedily hold of some, and leaving others in their immediate neighbourhood untouched!

That the skin disease and the nerve disease are due to primary independent elective tendencies on the part of the original disease, and are not secondary the one to the other, whether the skin condition to the nerve disease, or the nerve condition to the skin disease (both of which statements have been made), can I think be proved by a reference to cases. A typical case of tuberculated leprosy is non-anæsthetic. Dr. Hillis lays stress upon this absence of anæsthesia in his general description of this variety of the disease. He says: "the palm of the hand also becomes fissured, horny and scaly from disease or malnutrition, *but not anæsthetic*" (the italics are his). Further on he says: "Anæsthesia ensues when the pressure of the blastema is more than ordinary," proving that it is only in exceptional cases of great thickening of the skin that it is met with.

Thus we see that the nerves are generally unaffected in those cases where the skin condition is most marked. On the other hand, in the typical anæsthetic form, though the skin is rarely quite free from deposit, yet it is present in comparatively small amount. Neither of these propositions could be true if there was any causal connection between the tuberculated skin condition and the nerve condition giving rise to the anæsthesia. But the independence of the two conditions is best seen in mixed cases of nerve and skin disease, as in the patient whose case I have narrated. In him the skin condition began on the trunk, and the patches of affected skin did not become anæsthetic; whilst on the other hand the nerve condition began at the extremities of the limbs, and, if we trust his history, independently of any skin disease. I think

therefore we may take it as proved, that the skin and the nerves constitute two independent seats of election for the original deposit.

There is, however, one way in which the skin will be liable to be affected by the nerve disease. It is well known how large an influence the nervous system has on the nutrition of tissues of all sorts; and in proof of its influence on the skin, we have only to recall the conditions induced when a nerve has been permanently severed. The skin becomes degraded in every way; it atrophies, its glands cease to secrete, and great care has to be exercised to prevent it from breaking down into low forms of ulceration. The same peripheral nerve-lesion, therefore, which causes the anæsthesia of leprosy, will cause atrophy of the skin, such as we saw had taken place in Mr. D. But if this be so with the healthy tissues of the skin, so will it almost certainly be the same with morbid growths, especially if of sluggish nature, like that of leprosy. Hence, supposing that a patch of skin were invaded by the morbid growth at the same time as the nerve supplying the patch was also attacked, the death of the nerve would lead to cessation of the skin growth. From this it will result, that the difference between tuberculated and anæsthetic leprosy will become accentuated; for, in the former case, there is nothing to prevent the extensive deposit of the leprous neoplasm in the skin, whereas in the latter, even though there may be just as strong a tendency to the invasion of the skin, the simultaneous invasion of the nerves will hinder, and indeed almost prohibit, the skin affection in areas controlled by those nerves.

I will close my paper by suggesting a few points for investigation in future cases.

(1.) In mixed cases of tuberculated and anæsthetic leprosy, to notice accurately the difference between the eruption of parts of the skin where sensation is healthy, and that of patches of anæsthetic skin. With a view to proving the secondary influence of nerve disease in checking the morbid growth in the skin.

(2.) Is anæsthesia always preceded by modification in skin nutrition? With a view to proving the independence of the nerve disease.

(3.) Does the primary invasion of anæsthesia ever give rise to patches of numbness corresponding to the supply of any named nerve? With a view to clearing up the question whether nerve-trunks are ever subjected to direct attack.

(4.) Does muscular atrophy ever take place without being preceded by anæsthesia? With a view to proving whether ultimate motor branches may be primarily affected, in a way similar to ultimate sensory branches.

(5.) Where muscular atrophy has taken place, to make a detailed examination of the various group of muscles. With a view to confirming or confuting the conclusions arrived at above, in reference to the mode in which their atrophy is produced.

(6.) An investigation of the deep sensibility of parts beneath anæsthetic skin, and a comparison of the deep sensibility of healthy muscles as compared with that of atrophied muscles.

GENERAL PARALYSIS OF THE INSANE. A STUDY OF THE DEEP REFLEXES, AND PATHOLOGICAL CONDITION OF THE SPINAL CORD.

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THE study of the reflexes as a clinical symptom in nervous diseases, has of late commanded great attention. In 1875, Westphal and Erb each devoted a treatise to the study of this symptom, and the at present known ankle-clonus was then first described as the foot-phenomenon. Previous to this, Charcot, in his lectures at La Salpêtrière, pointed out in cases of paresis, or paralysis, of the lower extremities a certain trembling or trepidation, produced in the foot when it was caught by the point and suddenly turned back; since the treatise of Westphal, the foot-phenomenon has been carefully studied by various writers.

The study of the deep reflexes in General Paralysis of the Insane is exciting great interest among a small body of workers, their presence or absence tending to show that there is a close connection between general paralysis and other diseases, such as tabes dorsalis, and affording to some extent a basis for classification of a disease, which at present includes in a common description forms differing clinically and pathologically. Of the deep or so-called tendon-reflexes, the knee-phenomenon or patellar-tendon reflex, and the foot-phenomenon, or ankle-clonus, have been the most carefully studied. The purely reflex character of these phenomena is, however, at present strongly contested; from experiments and physiological data Westphal, Waller,[1] and others, conclude that the phenomenon is a local one. Be these opinions correct or not, the interesting fact remains,—that whatever increases or diminishes reflex action generally, increases or diminishes

[1] See 'BRAIN,' 1880.

the phenomenon under consideration. So far as our present knowledge extends, the following are among the most important causes which produce exaggerated and diminished reflexes.

(*a.*) Exaggerated reflexes.

1. Increased irritability of any factor of the reflex loop.
2. By far the most important condition, where cerebral influence is withdrawn from the spinal cord, by disease of some portion of the pyramidal tract, whether at the origin of those fibres in the cortex of the brain, in their passage through the corona radiata, internal capsule, pons, medulla oblongata, or in the spinal cord itself.

(*b.*) Diminished or abolished reflexes.

1. In tabes dorsalis, where the lesion is situated in the reflex loop, involving the fibres of the internal bundle of the posterior roots, or what Charcot calls "the inner radicular fasciculus."
2. In disease of the anterior grey horns, as in infantile paralysis, and in the advanced stage of pseudo-hypertrophic paralysis.
3. In disease of the posterior nerve roots.

By far the most important of these causes of changed reflexes are those:—1st, where there is lateral sclerosis of the spinal cord; this condition, it has been shown, by cutting off the inhibitory influence of the brain, gives rise to an increased reflex. 2nd, where there is posterior sclerosis of the spinal cord; in this case the reflex loop is directly interfered with. It thus follows that in considering the reflexes in any disease, one must at the same time take into account the pathology of the spinal cord. My own observations have been made with the following ends in view:—

1st. To determine the state of the reflexes in general paralysis.

2nd. To enquire into the connection between the reflexes and other clinical symptoms; and

3rd. To compare the state of the spinal cord after death with the state of the reflexes during life.

The following is an analysis of the condition of the deep reflexes in 65 cases of general paralysis which have come

under my notice; the observations were made at different times, and during different stages of disease, the results varying only in a way I shall subsequently describe. In some of the cases I am able to describe the pathological lesions found after death.

In the 65 cases examined, the knee-jerk was considered to be normal in 11, slightly exaggerated in 8, decidedly exaggerated in 18; it was diminished in 5, and absent in 18; in 5 cases the amount differed on the two sides. Ankle-clonus was absent in 47 of the cases, in one case it was not noted, and in 17 cases was present; being marked in 7, slight in 7, and in 3 cases present on one side and absent on the other. Of the 18 cases in which the knee-reflex is noted as decidedly exaggerated, 16 had ankle-clonus; the remaining case, in which ankle-clonus was present, had a slightly exaggerated knee-reflex. It thus appears that more than half of the cases examined showed marked alteration in the character of the tendon reflexes.

I will first consider those cases in which the knee-reflex, and with it ankle-clonus, was absent, omitting the cases where the knee-reflex was diminished, as I consider the question of diminution and slight exaggeration a personal equation. I am inclined to think that when the deep reflexes are absent, the mental symptoms are not so characteristic of general paralysis as in the opposite condition,—that of exaggerated reflexes; there may be delusions of an exalted character, but they are not so extravagant or obtrusive, and are, I think, apt to disappear early, leaving a more or less demented condition: in some this dementia is of the ordinary consecutive character; in others it is accompanied by a quiet expression of well-being and happiness, appearing in such expressions as, "I'm very well," "very happy," "as well as ever I was in my life," &c., generally attending this negative state, in an absence of that physical restlessness, which is, in my experience, a feature in those with increased reflexes. The patient is dull, sometimes sleepy, and will sit in one place for hours, staring vacantly in front of him. In common with other cases, they are subject to such accidents as apoplectiform and epileptiform attacks.

The state of the pupil demands closer attention; in the majority of cases they were unequal, and in many irregular; in a large majority of the cases accommodation appeared perfect, whilst the reflex to light was absent or sluggish; there was, in fact, the Argyll Robertson pupil; the pupils were usually contracted, but in some cases normal in size, or even dilated.

I find no uniformity in the condition of the superficial reflexes, they may be normal or diminished.

The absence of a knee-jerk suggests a connection between the cases of general paralysis, now under consideration, and tabes dorsalis, and I have looked for other symptoms of the latter disease; in some of the cases, inco-ordination of movements has been detected, it has not, however, been a prominent symptom, being, as I believe, masked by the state of general paresis. Cases have been described in which ordinary insanity has been associated with tabes dorsalis; the insanity, however, following on the ataxy. A case in point is one described by Dr. Newcombe.[1] In this case, the ataxic preceded the mental symptoms by more than two years; in my cases, ataxic symptoms were only apparent late in the disease, and were never prominent. Cases, however, have been described in which the ataxia preceded the general paralysis.

The frequent presence of spinal myosis in general paralysis is an interesting symptom, common to it and to tabes dorsalis, and optic disc atrophy has been frequently observed in both diseases; it was present in some of my cases of the former disease. I have never observed skin eruptions, gastric crises, cephalalgia, diplopia, or marked anæsthesia of cerebral nerves. I have also enquired for the lightning pains of tabes dorsalis; and I may note that a case has recently been published by my late colleague, Dr. T. S. Sheldon,[2] in which he says, "the patient suffered from peculiar pains about the waist, and in the legs, which he described by darting his fingers rapidly forwards." Notwithstanding these negative observations, the absent knee-jerk points to an organic connection between the two diseases, and this indication has been confirmed in three cases in which

[1] 'Brain,' Vol. II. p. 134.

[2] 'Bristol Medico-Chirurgical Journal,' December, 1884.

I made post-mortem examinations, finding sclerosis of the posterior columns of the spinal cord, diagnosed during life by the absence of the knee-jerk.

Case I.—John P., aged 55 years, a labourer, formerly a soldier, was admitted in January, 1883. Previous history negative, drank hard occasionally. On admission, patient is a small thin man, is somewhat excited. He has delusions of persecution and ill-treatment in the workhouse, saying the attendants kicked him to pieces, smashed his windpipe, so that he has not been able to speak properly since; also says he was scalped while in the Indian Mutiny. The pupils are irregular and unequal, the right being the larger; tongue protrudes slightly to the left, but is fairly steady. Delusion of his own greatness while in the army continued, and a general paresis of movements became evident.

In December he had an apoplectiform attack, the legs being paralysed, the right appearing the worse. Three days afterwards, the general paresis was found to have increased, the right arm being weaker than the left. The pupils contracted and unequal; the deep reflexes were completely absent.

In January, 1884, the pupils are found to react but slightly to light, and actively to accommodation; he has great difficulty in co-ordinating his movements, and cannot stand with his eyes shut; he resists ophthalmoscopic examination. He has delusions of putrefaction; his mental condition is now one of dementia.

March.—He has had two attacks similar to above, becoming more helpless and paralysed; after this he deteriorated rapidly, becoming bedridden, and died on March 12.

Autopsy (twelve hours after death).—Body fairly nourished. Brain 43½ oz.; the cranial cavity contained a quantity of fluid, vessels uniformly thickened, but free from atheroma. External convex surface of hemispheres affected to an extreme degree; wasting of the left hemisphere very marked; the meninges covering the frontal lobes and motor area are of a milky opacity.

Right hemisphere.—Meninges strip without adhesion from whole of convex surface, the gyri are atrophied, and diagrammatic in arrangement; they are much plumper than on the left side.

Left hemisphere.—Opacity and thickening of meninges marked to an extreme degree. Over fore-part of brain there are numerous small depressions filled with subarachnoid fluid; no adhesions over surface. Island of Reil much smaller on left than on right side. Pons wasted, crura small.

Spinal Cord.—To the naked eye the cord is tough and wasted;

there is distinct sclerosis of both posterior columns, all through the cord, and extending into the medulla oblongata. The pia mater over posterior columns is distinctly thickened. On section, the posterior columns are tough and gristly. Examined microscopically, the sclerosed tracts occupy the following positions. In the cervical region it is limited chiefly to the inner radicular zone. In the dorsal region it is a narrow line running from the centre of inner radicular zone, through the columns of Goll; and in the lumbar region, where the sclerosis is most extensive, it involves chiefly the inner radicular zone.

Case II.—Joseph P., aged 32, a shoemaker. Admitted Nov. 6th, 1882. On admission, his condition was one of dementia, with excitement. He is said to have had delusions of wealth for some time, and to have been wet and dirty in habits. He is very demented, and unable to answer simple questions. The tongue is protruded in a very tremulous manner. The pupils are unequal in size, the right is ovoid in shape; his aspect is sullen. Locomotion is good. He is dirty in habits, drinking his own urine.

In *December*.—His speech is embarrassed, thick, blurred, and occasionally stuttering; he answers questions simply but coherently; he walks fairly well, but general tremor is very evident; he cannot write a connected letter. The deep reflexes are entirely absent.

January, 1883.—His dementia and general paralysis have very much increased, his articulation is worse. Is in bed with suppurating sores at all points of contact with bed-clothes.

His condition got rapidly worse, and he died on the 4th of February.

Autopsy (nineteen hours after death).—Body fairly nourished, covered with bedsores. Calvarium thick, dense and white.

Brain 43 oz. on first removal; after removing membranes and draining fluid off, it weighed 36½ oz. There is much thickening and opacity of pia-arachnoid over frontal and parietal lobes, occipital portion fairly healthy; the arachnoid contains a quantity of brownish fluid.

Right hemisphere.—All the convolutions are extremely wasted, but especially the ascending parietal, and parietal lobules.

Left hemisphere.—Also wasted; the 1st, 2nd, and 3rd frontals most affected. The right temporo-sphenoidal lobe is soft and wasted. The membranes are adherent in one or two places to the parietal lobules on the right, and to the second convolution of the right temporo-sphenoidal lobe.

On section, cortex in frontal region very thin, lenticular

nuclei extremely small, ventricles much dilated, other parts wasted, brain œdematous. The floor of 4th ventricle was finely granular.

Spinal cord.—The cord is small, of fair consistence; no naked-eye changes were noted through whole of white columns of cord, membranes were not thickened. On microscopical examination, there is seen to be a marked sclerosis of the posterior columns, and other changes.

In lumbar region, the sclerosis involves the inner radicular fibres, extending thence downwards and inwards, occupying about the centre of Goll's column; it does not extend to the surface of cord, or up to the commissure; and a narrow tract bordering the posterior fissure is also unaffected; the lesion is most extensive in this region.

In the dorsal region, the sclerosis extends from the middle of the inner margin of the posterior horn, for a short distance downwards, occupying mainly the outer portion of Burdach's column, Goll's column being almost free. Under a high power the sclerosed tract shows nerve fibres varying in diameter, widely separated by a network of connective tissue. There is thickening of the vessels in the cord, and the following is the condition of a vessel running down the anterior fissure in the upper lumbar region: the wall is much thickened; there is a hyaline fibroid appearance, and the calibre is small. The inner layer of the pia mater is occupied by an almost homogeneous transparent material, which at certain points pushes itself amongst the nerve-fibres, towards which it presents a scolloped appearance.

In sections from this region, the pia mater bordering a portion of the lateral columns of the cord is much thickened, and translucent in appearance, it has a faintly granular look with numerous highly and refracting circular bodies scattered about in it.

Case III.—This case presented the symptoms which I have noted as characteristic of absent reflexes, and the spinal cord, examined microscopically, presented the following lesion. There is a marked sclerosis of posterior columns through whole of cord, being chiefly limited to the inner radicular fibres; but in the lumbar region, nearly the whole of posterior columns appear to be involved.

Three cases are of course insufficient to prove a rule, still it is an interesting fact that, the knee-jerk being absent during life, sclerosis of the posterior columns and other

changes noted in the spinal cord, should have been discovered after death, and I am inclined to believe that this lesion will be found in most, if not in all, cases of general paralysis, in which the knee-jerk is markedly absent. We must remember that there are cases of one-sided-brain disease, in which the knee-jerk is absent, and in which there is no lesion of the cord. Such a case is published by Dr. Mackenzie in 'BRAIN,' Vol. VI. p. 222, but such lesions as would lead to this result, may be excluded from general paralysis, the cortex lesion in which disease would rather suggest a descending degeneration. Why posterior sclerosis, an ascending lesion as we are taught, should be associated with general paralysis, is a question I am not prepared to answer; that it is not a mere coincidence may be inferred from the large proportion of cases in which the knee-reflex was found absent, and from the direct evidence of other histologists.

One of the most interesting observations on this subject is by Dr. Dowse:[1] he draws attention to some of the disturbances of reflex action common to general paralysis of the insane, and to tabes dorsalis. He says, "the patellar tendon-reflex may be absent in both diseases," and he is inclined to believe when such is the case in general paralysis, some change in the posterior columns of the cord has occurred. He gives Westphal's view of pathology, namely, that no direct relation exists between the morbid process in the cord and the morbid process in the brain, but that they are the expression of an excessive proclivity to disease of the nervous system. Westphal describes a sclerosis of the posterior columns similar to that in the cases I have related, and designates such as the "tabetic form" of general paralysis, a tabic gait being present. In my experience this form of gait is present in some cases. It was marked in the case of John P., Case I., but was not at all marked in the other cases, and its absence has been noted by other observers.

Exaggeration of the patellar tendon-reflex, associated in a large number of the cases with ankle-clonus, was found in twenty-six of the sixty-five cases examined. The ankle-clonus was marked in some of the cases, and slight in others; and in

[1] 'Brit. Med. Journ.' vol. i. 1882.

the cases in which it was present, varied considerably in amount at different periods, being at one time easily obtained, and at others with difficulty, or not at all. In my experience, the cases which present these exaggerated reflexes are the most typical examples of the disease, corresponding much more nearly with the classical description than do those in which the reflexes are absent. The delusions of grandeur are more constant and obtrusive; they possess great wealth, can perform great deeds, devise plans of an impossible and gigantic character, and invest mean surroundings with charms that do not exist. They are usually loquacious, and restless in their movements; there is a notable impairment of the finer muscular actions—those of the mouth, lips, and fingers—and in speech they slur their words. In the earliest stages there is a peculiarity in the gait of marked cases, the step being elastic and springy, and they are constantly on the move. In shaking hands, the act is exaggerated, the grip being convulsively firm. Savage has noted a tendency to fix the eyebrows, before they begin to speak, and in one case under my care this was well marked. These cases show tendency to fat-formation; the face becomes bloated and expressionless; ultimately there may be a rapid loss of flesh, and the paralysis becomes more marked; generally there is a difference in the amount of paralysis of the two sides. The condition of the pupils is not constant; in many there is irregularity of outline, and in most cases inequality; and in a few cases loss of reaction to light, whilst accommodation remains. Contrary to expectation, Savage has found in these cases optic-disc atrophy. The presence of an exaggerated deep reflex is not so valuable a sign of spinal-cord lesion as is the absence. Some observers state that both increased patellar reflex and ankle-clonus can be present without such lesion, as is the case sometimes in hysterical paraplegia.

The patellar reflex, being a phenomenon usually visible in health, and varying in amount in different individuals, there is always some difficulty in determining an abnormal amount. When ankle-clonus, however, accompanies increased patellar reflex, it is no doubt a grave reason for suspecting sclerosis of the pyramidal tract, more especially in such a disease as

general paralysis, in which disease of the cortex of the motor convolutions is often a marked feature. In the case of general paralysis, an interesting question arises as to the pathology of sclerosis of the pyramidal tract; as a rule, interruption of the tract at some given point, by injury or disease, starts the descending degeneration. Where does this interruption take place in general paralysis? In the case of G. J. F. (Case IV.) there was a double lateral sclerosis; but the most careful search failed to make out any break of continuity in the pyramidal tract. The meninges were not adherent to the cortex, but there was marked atrophy of the convolutions in Ferrier's motor area, from a portion of which, we are taught, the tract originates.

It seems to me that there are but two ways of explaining the presence of lateral sclerosis in such cases.

1st. That the atrophy of the pyramidal cells, probably the larger ones in the third layer of the cerebral cortex, is sufficient to give rise to a descending degeneration of the pyramidal fibres connected with them; in this connection I quote from Charcot, 'On Localization of Cerebral and Spinal Disease:' "It is not yet proved that destructive lesions, limited to the grey matter of the Rolandic region, are productive of secondary degenerations, nevertheless some facts taken from the pathological anatomy of general paralysis tend to show that this is really the case."

2nd. That, as explained when treating of posterior sclerosis, the sclerosis of the lateral columns may be part and parcel of a general increase of fibrous tissue in the cerebro-spinal axis.

I see no difficulty in the latter view, if we may believe in the existence of a primary lateral sclerosis. Westphal believes that in general paralysis only has primary lateral sclerosis yet been described; but Flechsig interprets the sclerosis as consecutive to cortical lesions. I am inclined to accept Flechsig's views, as lateral sclerosis occurs too frequently in general paralysis to be regarded as a mere coincidence.

Case IV.—G. J. F. Admitted September 1882. In January, 1881, he had a paralytic attack, from which he has never completely recovered; since then his mind has gradually become affected. His wife says he was walking home during a snowstorm when first attack occurred. After two months he had a second attack, on recommencing work, and has had two or three attacks since. With each attack he loses power of speech, and in left arm and leg. He has had delusions at times.

On admission.—He has a wild vacant expression, is quite incoherent, and cannot answer simple questions. Speech drawling and thick; tongue protrudes with a jerk; pupils unequal and irregular; there is much want of power in left arm and leg. Tendon reflexes much exaggerated, more particularly in left side. There is slight external strabismus of left eye.

October.—Is failing fast; general paresis increased; cannot stand; tendency to bed-sores.

Nov. 21.—Is in bed, both legs paralysed, but he moves them a little; uses arms apparently equally. As legs lie flat on the bed, a tap of right patellar tendon causes contraction of quadriceps muscle, similar to that produced by tapping the muscle itself. On left side a convulsive kick of limb follows. Sole reflexes seem normal and equal. Sensation is certainly deficient. Patient more demented.

Jan. 24, 1883.—Has gradually got more paralysed and weaker. This morning had right-sided convulsions; at noon there is no convulsion, but firm contraction in flexors of right arm; eyes turned to the left. Patient unconscious.

Jan. 28.—Died from pneumonia of left lung.

Autopsy.—Calvarium thickened. There is a bluish opacity and thickness of the basal vessels. Brain 50½ oz., very œdematous; pia-arachnoid thick and opaque over frontal lobes and motor area; the gyri are much wasted on both sides. No coarse lesion discovered on section.

Spinal cord.—To the naked eye there is marked sclerosis of both lateral columns, extending through whole of cord. Examined microscopically, the sclerosis occupies the usual position in the lateral columns, viz. what Charcot calls, the "crossed pyramidal fasciculi."

Case V.—J. V., aged 36, married. A builder's accountant.

Admitted Oct. 8th, 1883. Supposed causes, heredity and business failure. First symptoms, noticed six months ago, being loss of memory and mistakes in his work.

On admission.—Is a well-built, intelligent-looking man of medium height; has a self-satisfied expression. He talks coherently

and volubly; his memory is good. He has the most extravagant delusions; says he is the richest and most wonderful man living, that he has built palaces of diamonds, and invented a machine for paving the streets with gold at one penny a foot, &c. In conversation he occasionally stammers and slurs his words; has great difficulty in pronouncing such words as "statistical." His tongue and lips are tremulous, and he protrudes his tongue with a sudden jerk. There is capillary congestion over both malar bones, and in speaking he frequently fixes his eyebrows. In walking, his gait is springy and firm, he takes long strides, and walks a good deal on the toes. In shaking hands, he grips the hand firmly and convulsively. There is evident paresis of the fine muscular movements; his finger movements are awkward; he is a fair musician, and when playing the piano, he leaves out notes and slurs them. His pupils are unequal, the left being the larger; they react to both light and accommodation. His patellar tendon-reflexes are both markedly exaggerated, and there is ankle-clonus on both sides.

May 1884.—Has the same exalted delusions. The general paralysis has increased; knee-reflexes still exaggerated, but ankle-clonus obtained only with difficulty. He is extremely restless; constantly on the move; in walking droops left shoulder, and drags left foot slightly.

July.—Bodily weight has increased. Still has exalted delusions, but is quieter; does not talk so much; is irritable, and easily displeased.

Sept.—Is getting feeble, losing flesh. Mental condition more one of dementia. Knee-reflexes still exaggerated; ankle-clonus absent.

Here, unfortunately, I lost sight of the case; but I am informed by my late superintendent, Dr. A. Law Wade, that he died in January 1884, after a large number of epileptiform convulsions, chiefly on right side. The brain showed the usual lesions of general paralysis, and the spinal cord showed a later posterior sclerosis in lower dorsal region, with an enormously dilated central canal.

Cases VI. and VII.—These cases showed symptoms which I have previously noted as characteristic of an exaggerated deep reflex. I will therefore only give the condition of spinal cord, found after death.

Case VI.—G. C. To the naked eye, there is a marked sclerosis of both lateral columns in the usual positions; the cord is firm and tough. Examined microscopically, the sclerosis is visible through

extent of cord, but chiefly in dorsal region. Here and there are separate plates of connective tissue, and in one series of sections in the dorsal region there is a patch in posterior column on right side.

Case VII.—J. S. Cord firm, no wasting. To the naked eye, there is sclerosis of lateral columns in usual position; a fresh section shows same condition.

So far, therefore, as my observations have gone, it would appear that in the state of the deep reflexes we have valuable indications of the state of the cord in general paralysis of the insane. They also show that disease of the spinal cord exists far more frequently than has hitherto been understood; that in some cases the morbid process may commence in the brain, and in others terminate there.

A provisional classification can be made from the state of the deep reflexes as follows:—

1st. A tabic form, in which there is posterior sclerosis of spinal cord. 2nd. The paralytic form, in which there is lateral sclerosis of the spinal cord; and, 3rdly. A form which is cerebral rather than cerebro-spinal, physiological tracts in the cord not being affected.

Of the latter class, in which the reflexes are not altered, I have made post-mortems in eight cases. In none of these did I find a fasciculated sclerosis; the general symptoms follow more closely the classical description of the disease.

Clinical Cases.

CASE OF BRACHIAL MONOPLEGIA, DUE TO LESION OF THE INTERNAL CAPSULE.

BY A. HUGHES BENNETT, M.D., AND C. M. CAMPBELL, M.D.

General P., æt. 80, served his full time in India, but had retired, and lived in Europe for upwards of twenty years.

According to the statements of his relatives, his temper and disposition had changed for the worse during the past few years, —but his general health had been good. He suffered, however, from marked muscular tremors of both superior extremities, for an uncertain period, before he came under observation.

On November 17th, 1884, he dressed himself, went through several corridors, and descended in the lift to the saloon of the hotel in which he was staying.

When first seen, he was seated at breakfast, making partially successful attempts to feed himself.

He complained of difficulty of speech; and had remarked on getting up in the morning that his mouth was drawn to the right side. There was marked paresis of the left face and tongue, but no other part of the body was affected. The patient was ordered to go to bed, and avoid stimulants and excitement of any sort.

When next seen at 6 P.M., it was stated that an hour previously he had got up, and fallen insensible on the floor. He was now partially conscious; but the paresis of the left face was more marked, his speech was quite unintelligible, and voluntary motion of the left upper extremity entirely gone.

For some days, deglutition was so imperfect that the patient was chiefly fed per rectum. Urine was occasionally passed involuntarily, and the power of the sphincter ani was diminished.

On December 1st, articulation and deglutition were much improved, and facial paresis less marked. The movements of the eyeballs, vision, and the special senses were normal. The power of walking was not tested, but when in bed, no loss of power in the lower extremities could be detected. The knee-jerk was slightly more marked on the left side.

Subsequent examination showed the face to be normal. Although locomotion was feeble, there was no evidence of special paresis of the left leg. The arm remained as before, completely paralysed,—the muscles flaccid and without trace of rigidity. The sensibility was everywhere normal.

For about four weeks the general condition improved. About this period intelligence became clouded; the patient talked Hindustanee, or Italian, without apparently being aware that those about him did not understand him; he became lethargic, and unable to fix his attention. This condition continued to intensify, until he died on January 20th, 1885, without developing any new special symptoms.

Post-mortem Examination.—Permission was obtained, on the 21st, to examine the brain only. The dura mater was found thickened throughout, and in many places strongly adherent to the skull; the arachnoid and pia mater were deeply congested, opaque, and thickened, and there was considerable sub-arachnoid effusion. All the arteries noted were thickened and atheromatous. The convolutions generally were flattened, and soft to touch, but otherwise normal.

A series of vertical sections was made across the brain, which showed its substance to be slightly congested, and softened, but otherwise normal, except at the spot about to be described.

A vertical section, in the direction of the fissure of Rolando, through the middle of the right ascending parietal convolution, disclosed a limited defined softening, at the upper part of the internal capsule, or foot of the corona radiata. The softened area was about the size and shape of a horse-bean, though flatter, and occupied the position indicated in the accompanying diagram (Fig. 1, p. 80). It measured $\frac{5}{8}$ of an inch in its long, and $\frac{3}{8}$ of an inch in its short axis, but not more than $\frac{1}{8}$ of an inch in thickness antero-posteriorly. Its upper edge just touched and slightly involved the outer and upper border of the caudate nucleus. From thence it stretched obliquely downwards, across the internal capsule, to the upper margin of the lenticular nucleus, the apex of which it very slightly invaded. The caudate and lenticular nuclei, and the neighbourhood, were otherwise normal.

Commentary.—All the circumstances of the case during the life of the patient led to the assumption, that the lesion existed in the middle parietal fasciculus of the centrum ovale; in the triangle of white conducting substance, the base of which is the cortical substance of the ascending parietal convolution, and the apex, the upper part of the internal

capsule behind its knee. There was no evidence as to the exact level at which the interruption occurred.

After death, a limited, flat, and perfectly circumscribed softening was found occupying the upper part of the internal capsule, extending between the caudate and lenticular nuclei; its situation corresponding to a line of vertical section through the middle, and in the direction of the ascending parietal convolution. This had caused complete, universal, and permanent paralysis of the left superior extremity only; the antecedent and accompanying immobility of the left face being transitory, and weakness of the left leg, if it existed at

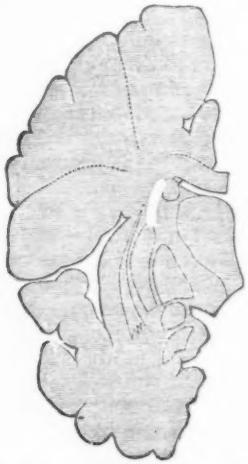


FIG. 1.—Vertical section of Hemisphere through the middle of the parietal convolution. The white patch marks area of softening between caudate and lenticular nuclei.

all, being equally so, as none was observed four weeks after the attack. The chief interest of the case lies in these phenomena, which differ from those of common general hemiplegia, due to more diffuse disease of this region. In all such cases, including the present one, the destruction of tissue is probably due either to hæmorrhage from, or to obstruction of a branch of the lenticulo-striate artery. But in the present instance the symptoms, at first somewhat diffused, doubtless from shock, subsequently remained strictly limited to the arm, the result of the flat patch of softening already described, stretching across the capsule in a nearly transverse direction.

The fact that such a lesion should cause permanent paralysis of the left superior extremity only, leaving the left face and leg unimpaired, shows that the fibres conducting motor impulses from the cerebral cortex run in separate ribbon-shaped bundles, through the internal capsule; and that their flattened surfaces are at right angles to an antero-posterior section of the cerebrum.

Assuming the same conditions for the other motor centres of the body, we may hazard a general idea of the arrangement of

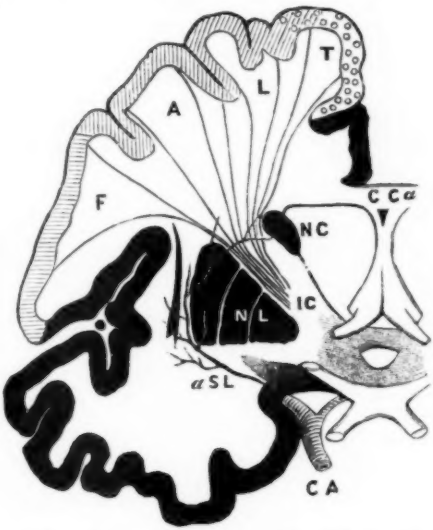


FIG. 2.—(After Horsley.) Vertical section of Hemisphere through the Fissure of Rolando, showing relative position of motor centres, F. Face, A. Arm, L. Leg, T. Trunk. The fibres run from motor centres into internal capsule between the nuclei of the corpus striatum and the "artery of hæmorrhage" (Duret). C. Ca. Corpus Callosum. N. C. Caudate nucleus; N. L. Lenticular nucleus. C. A. Internal Carotid Artery; a S. L. Lenticulo-Striate Artery. Cf. fig. page 132.

the conducting fibres, between the cortex of the brain, through the pyramidal tract to the internal capsule. Round the fissure of Rolando are situated those centres which preside over the voluntary movements of the trunk, leg, arm and face of the opposite side of the body. This sulcus lying in an oblique direction, downwards, forwards, and outwards, makes it evident that the various areas lie not only at different levels, but in different positions as regards their

antero-posterior and lateral situations. Thus the centre for the trunk is the most superior, posterior, and internal. Immediately contiguous is the centre for the leg; next in order, that for the arm; next and last, that for the face, which is the most inferior anterior and external. The four bundles of conducting fibres, descending from these centres in the above order and position, in a downward and obliquely inward course, may be compared, when viewed from the front, to four rays of a half-opened fan. These converging in this attitude toward the upper part of the internal capsule enter its knee, and as they do so, change their direction. Preserving their

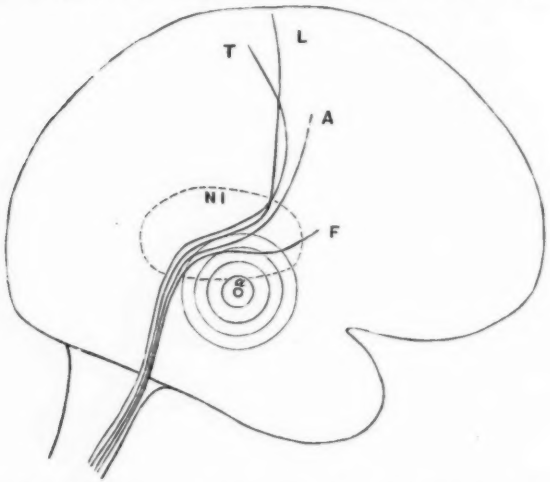


FIG. 3.—(After Horsley.) Motor centres as seen from the side. F. A. L. T, as in Fig. 2. N. I. Lenticular nucleus, indicated by the dotted line. The fibres of the direct pyramidal tract are seen passing down to the internal capsule, arranged first in a superior-inferior order, and afterwards in an antero-posterior order.

relative stations, they become twisted from their former oblique position, to one completely antero-posterior. So that what was external and in front, becomes anterior; and what was internal and behind, becomes posterior. The half-opened fan is now shut, and its rays, which before were seen obliquely in front, are now seen from the side. In this last position, all the series of fibres seem to pass through the internal capsule in well-defined ribbon-shaped bundles, those for the face being the most anterior, behind which in successive order we find those for the arm, leg and trunk

That some such arrangement existed, had already been suggested, especially in a recent paper by Mr. Victor Horsley on "Substitution"[1] The present case supports these hypothetical views, by showing that a limited, flat, transverse lesion of the internal capsule can cause paralysis of the superior extremity, without destroying the mobility of the face and inferior extremity. In short, the second anterior ray of the

FIG. 4.—Horizontal section through Basal Ganglia, showing the entire horizontal length of the internal capsule. The white patch on the right side behind the knee represents the probable situation of the softening, the other aspect of which is seen in Fig. 1.

fan may be divided and destroyed, those in front of, and behind it, remaining intact.

It would have been a point of interest to have demonstrated the exact antero-posterior position in the internal capsule which this limited lesion occupied. The whole brain, however,

[1] 'Lancet,' July 5, 1884. See also Reviews and Abstracts in this No. of 'BRAIN.'

could not be obtained for further investigation, as permission for the autopsy was only granted on condition that the organs should be returned. A small section containing the lesion was however retained, which establishes and preserves the size and situation of the lesion, as demonstrated by a *vertical* section; but its exact position, on *transverse* section, could not be obtained.

The following experiment was therefore made on a normal brain. A vertical section having been made in the same position as that in the specimen, viz. down the centre of the ascending parietal convolution in the direction of the fissure of Rolando, and a transverse section through the middle of the internal capsule, through the basal ganglia, it was found that the spot at which they bisected each other, was a little behind its knee (Fig. 2).

This is where the lesion, presumably, would have been found.

AN UNUSUAL CASE OF ATHETOSIS.

BY SEYMOUR J. SHARKEY, M.B.,

Assistant Physician and Joint Lecturer on Pathology at St. Thomas's Hospital.

G., ÆT. 24, came under my care as an out-patient at St. Thomas's Hospital, on March 26th, 1884.

Until the patient was three years old he was quite healthy; he was then suddenly attacked with loss of power all down the left side, accompanied by repeated and prolonged convulsions.

As far as he knows, the movements of the limbs and face on the left side gradually followed the loss of power, and have gone on increasing ever since; at any rate, they have done so as long as he can remember. When between four and five years of age he was taken to Great Ormond Street Hospital, where his parents were told that the movements were so slight, that he would get over them.

About three years ago weakness and movements came on in the right leg, and he went to Guy's Hospital, where he was told that nothing could be done for him. He then consulted a homœopathic chemist, who gave him "strengthening medicine," and his right leg got strong again, and the movements ceased in three months.

A month ago the weakness and movements commenced again in the right leg, and they have gradually increased. The arm is said to be free from them.

The patient has never had any other severe illness, has not suffered from headaches, and has done his work well as a carpenter until the last six months. During this time the movements of the left hand have been so bad that he has been unable to hold the nails with accuracy, though he can hammer well enough with the right arm.

He says he has never had any defect of sight or hearing.

On rough examination, vision seems normal, and he hears the tick of a watch a long way off.

There are movements of the greater part of the muscles on the left side of the body, and they cease during sleep. There is no anæsthesia.

The tongue moves a little irregularly, as it does in chorea. Speech is drawling. The muscles both of the left and of the right side of face contract too strongly when the patient attempts to speak, so that a kind of grimace results, in which the lines at both angles of the mouth and the naso-labial furrows are very deeply marked.

These are spasmodic, more or less regular movements of the neck

to the left, and the shoulder is rhythmically raised and jerked slightly forwards and backwards.

One can also see the serrations of the left serratus magnus contracting rhythmically. The abdominal muscles are harder on the left side than on the right, but there are no evident movements in them.

There are slight spasmodic movements in the muscles of the left arm; but they are only slight. The hand, on the contrary, is markedly affected.

There is constant more or less rhythmical flexion and extension of the fingers, mainly at the metacarpo-phalangeal joints, with irregular flexion and extension of the phalangeal joints, and occasional separation of the fingers. In the left leg there are similar slight movements of the muscles, with marked movements of the toes, and most marked of the great toe. They are tremulous flexions and extensions of the toes; but the flexor longus hallucis is constantly contracted, so that the great toe is always riding over the others, and its proximal phalanx is usually at right angles to the metatarsal bone. In the right foot the movements are similar, but not so marked. In the right hand only the little finger moves, and that so slightly, that the patient had not noticed it. The grasp of the right hand, though stronger than that of the left, appears to be deficient in power; the patient says, however, that he has never noticed that it was weak. There is neither atrophy nor hypertrophy of the muscles of the limbs. The patellar reflex is unnaturally brisk on both sides, but there is no ankle-clonus.

The patient seems very intelligent, and says that his memory and mental power are very good.

Fundus of eyes and fields of vision natural.

Urine contains no albumen.

June 7th.—Admitted into St. Thomas's Hospital.

While an out-patient he had been treated with Easton's syrup, and subsequently with phosphorus in pills, but without any good result. Indeed the movements on the right side had rather increased.

On admission.—The right middle and ring, as well as the little finger, present rhythmical movements, and the wrist is turned slightly to the ulnar side. There are, in addition, occasional contractions of the right sterno-mastoid muscle.

The abdominal muscles are hard on both sides, though more so on the left than on the right, and the divisions of the recti can be clearly seen.

The patient sleeps badly, but he cannot say why.

While in hospital for several weeks he was treated with the continuous current and improved very much. The movements diminished decidedly on the right side, but never ceased altogether. The patient expressed himself as very much better and able to do many things which he formerly could not do. He was then sent away to the country.

Remarks.—Oulmont, in his Monograph on Athetosis, describes two forms of the affection, one unilateral, the other bilateral. The former, hemiathetosis, he says is an affection generally symptomatic of a cerebral lesion, and consists essentially of involuntary movements, habitually continuous, slow and exaggerated, and limited to the hand and foot of the paralysed side. Bilateral athetosis, on the other hand, he describes as an affection always primary, consisting essentially of involuntary, slow movements, involving the two hands, or the hands and feet together, and sometimes the two sides of the face. Several characters distinguish it from unilateral athetosis: first, its origin; for it is a primary affection of the earliest childhood, or perhaps congenital. Secondly, its localisation; for it involves the extremities of both sides of the body, and sometimes also both sides of the face. Finally, the movements are much more feeble than in hemiathetosis, often intermittent, and occurring only during attempts at voluntary movement. Moreover, it is usually associated with idiocy.

The case described in this paper must be placed in the same category as those described by Oulmont as hemiathetosis. For it was not congenital, but originated in a sudden attack of illness which resulted in hemiplegia: it remained for many years unilateral; and the intellectual capacities of the patient have never been in the least degree deficient.

The peculiarities of the case which seem to make it worthy of record are, first, the gradual supervention of movements in the muscles of the right side of the body after the disease had been confined for eighteen years to the left: and secondly, the great number of muscles which are affected in addition to those of the hand and foot.

Oulmont attributes the affection to disease of what he calls the "Athetosic fibres," which he supposes to exist in the posterior part of the internal capsule in front of and outside the sensory tract. Such a localisation of the disease appears to be very unlikely. It may, however, be presumed that in all probability the affection in the present case resulted from some coarse and sudden lesion, possibly hæmorrhage, in some portion of the right hemisphere situated more deeply than the cortex.

It would be exceedingly interesting to know by what means, or by what route, the movements spread to the opposite side, whether by means of the commissures of the brain, or by the direct pyramidal tract, or by fibres or cell-processes connecting the large motor cells of the anterior cornua of the spinal cord. But there do not appear to be sufficient data for determining this point; the case is therefore simply recorded, and speculation is left for the reader.

CASE OF PERFORATING TUBERCULOSIS OF SKULL WITH CEREBRAL SYMPTOMS.

BY WALTER EDMUNDS.

PATIENT was a boy, aged 14, of a rather delicate family, one of his sisters having died of hip disease.

Eighteen months before admission, he had an attack of peritonitis which was thought to be tubercular.

About the same time, there formed on the left side of the scalp over the parietal bone a cold abscess, accompanied by much headache; this abscess was twice aspirated, and thick pus drawn off each time.

Some six months later he complained, on and off of numbness in the right arm and hand, and four months later the abscess in the scalp began to fill again.

Two days before admission he had a fit—the first he had ever had. He felt giddy, fell, became unconscious, foamed at the mouth, bit his tongue, and passed water involuntarily; both legs were convulsed, and the right arm clenched and drawn up.

State on admission: A pale but well-nourished boy; on the left side of the scalp is a soft fluctuating tumour, from which a mixture of blood and pus can be drawn out with a fine syringe.

The grasp of the right hand is much weaker than that of the left; the right leg is also weaker than the left, but the difference is only small; there is, too, slight right facial paresis.

Sensation everywhere normal.

Ophthalmoscopically both discs are slightly swollen and their margins blurred; the veins kinked, and white lines along the vessels. No hæmorrhages.

Temperature normal. Sleeps well. No headache. No albumen or sugar in urine. He never vomited, and never noticed any defect of sight. Knee-reflexes normal.

Under ether the abscess was laid freely open. At its base was found a piece of diseased bone of oval shape, measuring seven-eighths by five-eighths of an inch; it was depressed on one side, and nearly completely separated from the surrounding bone, so that with an elevator it could be lifted up and removed.

It comprised the whole thickness of the skull, the dura mater being exposed, and the pulsations of the brain seen.

The wound was dressed with iodoform, and (except for the formation of a diminutive aneurism in the scalp which had to be opened) progressed uniformly well; there was, however, some suppuration with loss of substance on the surface of the brain.

He had two fits subsequently; one two weeks, the other eight weeks, after the operation; in both of them the head was turned to the right, he had general convulsions and foamed at the mouth.

The discharge from the openings in the skull gradually diminished, and the scalp covered over the edges of the skull,

(From a Photograph.) The lines indicate the great longitudinal fissure, the two branches of the fissure of Sylvius and the fissure of Rolando. The cross is at the parietal eminence.

so that when he was discharged there was an opening in the scalp and skull through which the brain could be seen. A silver plate was made to protect this.

The inflammation of the optic discs gradually subsided, but that of the right eye is not yet quite normal.

In about four months he was sufficiently well to get up and walk about; the chief thing remaining wrong with him, besides the opening in the skull, was the weakness of the arm and hand, the strength of the right hand being only a third of that of the left.

Shortly before he went out, I drew out on the boy's shaven scalp with the assistance of my friend Dr. Reid, the lines

he has given, for indicating as nearly as possible the positions of the cerebral fissures. (See woodcut.)

The opening in the skull was seen to be half-an-inch in front of the middle of the fissure of Rolando; but as the superficial ulceration of the brain extended for a short distance backwards from the opening, the middle of the ascending frontal and ascending parietal convolutions were affected. Probably the centre marked VIII. and one of the centres marked X. by Ferrier were involved; these are concerned, he says, with the flexion of the forearm and the movements of the hand and wrist.

This case is recorded partly as an example of a somewhat rare disease, "Perforating Tuberculosis of Skull," an affection which has been carefully described by Volkmann in the 'Centralblatt für Chirurgie,' 1880, No. 1, and partly because it shows a coincidence between the localising cerebral symptoms and the measurements on the surface of the scalp.

CASE OF PACHYMENINGITIS CERVICALIS HYPERTROPHICA.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.,

Physician to the Queen's Hospital, Birmingham.

W. B., a boy aged 18, was admitted into the Workhouse Infirmary, May 25th, 1883, with paralysis and tucking of both legs.

When five years of age, he fell down some steps on to the back of his head. Ever since, he has had an internal strabismus of the left eye. Another fall two years previously to his admission, he says, caused the paralysis; in his opinion the paralysis commenced shortly after.

The patient had been in the General Hospital for twelve months before he was brought to the infirmary, and while there, both arms and legs were paretic and rigid. The paralysis began in the right arm; the onset was gradual, and attended with numbness, tingling, and loss of sensation.

There was great pain in the neck and scalp, for which a seton was introduced, and the neck was rigid, movement causing great pain. The pain was along the great occipital and third cervical nerves, upward towards the head, also down the shoulders.

Two or three months after the paralysis of the right arm, the left arm, both legs and back, began to get weaker, until the patient was completely paralysed.

The arms were rigid, with extreme flexion of the fingers and wrists. There was no paralysis of the bladder or rectum.

Five months before admission (in 1883) he began to recover the use of his arms, and they have been getting better ever since.

There was never any facial paralysis, or any cerebral affection or disease of the cranial nerves, no vertigo, delirium, or vomiting.

The legs were weak when the arms were affected. At first they were flaccid, but they gradually began to get stiff, and contracted and trembled. All four extremities were paralysed together, but the arms got better while the legs got stiff.

When admitted, the patient was much emaciated, had spasmodic internal strabismus of the left eye and torticollis, the

head being twisted to the right. He could rotate his head more to the right than to the left.

No tenderness could be elicited by percussion over the cervical spines, and there was no fever or any sign of scrofula.

Both legs were paralysed and flexed at the hips and knees, with adduction of the thighs.

The arms had recovered power, but were still weak. There were no sensory disturbances.

Ankle-clonus and front tap contraction were markedly present in both legs. On account of the contracture, the patellar reflexes could not be obtained.

The plantar reflexes were present; the cremasteric, abdominal and epigastric absent.

There was no sensory disturbance in the lower extremities, and no marked wasting.

The optic disc and fundus on each side was normal. The patient was treated with iodide of potassium, and counter-irritated over the cervical spine, and when examined in July 1884, was much better, could rotate the head more, could use the right leg well, and there was little rigidity on this side, but still ankle-clonus.

The left leg was still tucked, and wasted with increase of reflexes.

He was well fed, and spent as much time in the open air as possible, going about on crutches. A shot bag was suspended to the ankle of the left foot, to diminish the contracture. On the 14th of March last, the patient, wishing to leave, was again examined. The chin was still slightly turned to the right, the reflexes (bicipital, &c.) in the upper extremities well marked, but there was no rigidity, and the grasp on both sides was very powerful.

Ankle-clonus had disappeared, and the contracture of the left leg much diminished. He could not, however, put the left heel to the ground, there being talipes equinus. The left leg was considerably wasted. The circumference of the left calf was 10½ inches, of the right, 13 inches.

This wasting can only be accounted for by supposing that an extension of the morbid process in the crossed pyramidal path to the anterior cornua has taken place.

The electrical reactions of the nerves and muscles of the left leg are normal.

As to the diagnosis of the case:—

Amyotrophic lateral sclerosis is excluded by the marked sensory disorders and progress of the case to recovery.

A transverse myelitis, by the great predominance of the irritative phenomena, and absence of anæsthesia, and complete paralysis of the leg preceding the rigidity. Progressive

muscular atrophy by the presence of rigidity and sensory disturbances.

Atrophy is said not to occur in the leg in this disease, but in this case it has occurred in the left leg. There is no doubt that the lesion was one of the spinal membranes, and to be certain of the diagnosis of pachymeningitis cervicalis hypertrophica, we must eliminate Potts' disease and tumour of the membranes.

Potts' disease is excluded by the absence of spinal tenderness, deformity, cachexia and fever, also by the absence of scrofula.

Tumour is more difficult to eliminate, but the gradual recovery excludes it, so that there can be little doubt about the case. A traumatic form of the disease has been described, and this case was probably due to injury.

The position of the hands shows the lesion to have been in the upper half of the cervical enlargement of the cord.

Reviews and Notices of Books.

Outlines of Psychology. By JAMES SULLY, M.A., &c. London, Longmans and Co., 1884. Demy 8vo., pp. 711.

An Introduction to Mental Philosophy on the Inductive Method. By J. D. MORELL, A.M., LL.D. London: Stewart and Co. Crown 8vo., pp. 390.

THERE is not much in this book that calls for notice in a Journal of Neurology. The problems of subjective psychology as distinguished from philosophy are treated fully and clearly, but that aspect of the subject which deals with nervous processes, and with the relation of these processes to the operations of mind, does not occupy a prominent position in the dissertation. Indeed, considering that the work is written with special reference to the theory of education, it is probable that its value from this point of view would have been enhanced by a more copious and more formal inclusion of the neurological side of the question. Education, as Mr. Sully insists with much force, is no longer believed to consist in merely stuffing the memory with words. The so-called education which begins with learning the Latin grammar by rote, and ends with making Latin verses by rule, is now recognised to be as unscientific as it is disgusting; and even some schoolmasters admit that the aim of education is not so much the acquirement of knowledge, as the training of faculty. Now the training of faculty is, on its physiological side, the development of the nervous system in certain directions. Great as is the importance of the purely mental or subjective aspect of the development, it is not intrinsically greater than that of the physiological aspect; and in view of the narrow foothold that it has in pedagogics, the need of a full and careful exposition of the physiology of the subject in a psychological work, written with special reference to education, is the greater. In treating of attention, Mr. Sully points out how "when once the fresh interest of a thing is exhausted, a further fixing of attention costs more and more effort; the mind soon wearies, and attention flags in spite of the utmost effort." From the introspective point of view this statement is complete; but its interest and its value would be greatly enhanced if the writer went on to give the physiological basis of this fatigue, increased effort and final failure. When it is explained that fatigue, partial or general, is the feeling that accompanies excessive expenditure of energy by nervous regions, locally or generally,

that increased effort means further expenditure by regions whose surplus has been spent, and whose reserve is liberated only by strong stimuli; and that exhaustion means the absence of any stimulus sufficiently powerful to unlock this reserve; the flagging and failure of attention acquire fresh interest. And when it is further pointed out that an exhausted nerve region, while it can be compelled to go on expending energy by increasing the magnitude of the stimulus, yet discharges at the expense of the integrity of its structure, and that this structure is more easily so damaged in the plastic tissues of youth than in the comparatively rigid fabric of the adult nervous system, a clear and strong light is thrown upon the danger of urging children to attend for a long time together to any one kind of occupation. Given plenty of food, and plenty of sleep, children can scarcely be too fully occupied in their waking hours, but the occupation should be very frequently varied, and varied through the widest possible range of difference. It is scarcely fair to blame Mr. Sully for omitting a subject which he does not undertake to deal with, and it is quite open to him to retort upon neurologists the imperfections and shortcomings of their own science. It is quite true that we cannot yet boast of having settled, even among ourselves, the nature of the nervous process that underlies every mental state, but still, having regard to the great importance of the working of the nervous system from the educational point of view, and to the small amount of attention that has hitherto been paid to this matter by pedagogues, it is very desirable that a psychologist of Mr. Sully's eminence and weight should help to bring it into prominence.

Dr. Morell's work is a sound and useful text-book for students, adapted specially to the examinations of the University of London, and enriched with a large number of examination papers which enhance its usefulness. It is concisely and clearly written, and well adapted to the purpose in view. The neurological aspect of the subject is not dealt with, except incidentally in the opening chapters; and here it is surprising to find a positive assertion of the existence not only of "vital force," but of "force of mind," which, we are told, includes Will. "These three forces (vital force, nerve force, and mind force) stand in the closest correlation." This is a position that no neurologist can accept, and if it were of importance with reference to the remainder of the book, it would be impossible to do otherwise than condemn the latter; but so long as subjective processes alone are dealt with, the book is reliable.

CHARLES MERCIER.

On Sclerosis of the Spinal Cord, including Locomotor Ataxy and other System-diseases of the Spinal Cord. By JULIUS ALTHAUS, M.D. London: Longmans, 1885.

THE book of Dr. Althaus[1] consists mainly of a *résumé* of those facts which already were familiar to the initiated in the profession, more especially with reference to tabes dorsalis or locomotor ataxy. The reader versed in neurology will therefore not discover anything essentially new in it. The general practitioner, however, for whom, as the preface indicates, the book is chiefly intended, will not always be able to discover from its perusal to whose credit the discovery of certain facts is to be placed; and to him, therefore, the Author's share in it will often appear greater than is really the case. No doubt this is quite unintentional on the part of Dr. Althaus; but so many names are already quoted, that it would have been better either to abstain from any references at all, or to complete still further the list of authorities.

The anatomo-pathological part of the book is decidedly the weakest; one feels that the Author is not standing on a ground personally familiar to him. He would not otherwise commend the method of Bevan Lewis, which savours of an antiquated microscopical technique, referring, for instance, to the investigation of ganglionic cells by compressing them with the needle under the covering-glass.

The definition and conception of Spinal Sclerosis, with which the author opens this book, is very peculiar; and it is highly regrettable that it should be precisely introduced in a work intended for practitioners. It implies that in every case we have to deal with *systematised* disease of the cord, a statement which will strike the critical reader as questionable as well as novel. Dr. Althaus does not, however, experience any scruple in placing among systematic lesions *multiple sclerosis*, which has never been yet, and never will be reduced to that group.

Inaccuracies and positive errors occur in the detailed account of the anatomo-pathological conditions. The author has evidently read somewhere that in tabes no healthy fibres occur among the diseased tissues of the posterior roots and columns (p. 31). But whoever has ever seen for himself a section of the cord in tabes will have noticed that this is not the case.

The other diseases mentioned besides Locomotor Ataxy, such as Friedreich's disease and spastic paralysis, are treated far too briefly. Under the latter heading Dr. Althaus makes an original statement on the subject of the tendon-reflexes. He distinguishes three types, cerebral, spinal, muscular; the last is stated to occur in peripheral paralysis, in contradiction with all other observers, who are unanimous in recognising the entire absence of the deep reflexes in such cases.

[1] This review has been written from the author's German edition, 1884.

Amyotrophic Lateral Sclerosis is disposed of in *two* pages. We can hardly consider this space, nor that devoted to the account of multiple sclerosis, as at all sufficient.

With reference to the ætiology of tabes, Dr. Althaus declares himself to be on the side of those who recognise a causal relationship between it and syphilis, but forgets to mention the fact that in 1881, at the International Congress, he attacked this very same view. Cold and over-fatigue are also mentioned as causes.

We do not find in the account of the initial stage any new points worth noticing; but a case of labyrinthine affection, with tabiform symptoms in the legs of a transitory character, appears to us doubtful, as neither pupillary rigidity nor loss of knee-jerk are mentioned. We need not discuss the statement, that attacks of monoplegia and aphasia are not uncommon at the outset of tabes. On the whole, however, the phenomena observed in the initial stage are carefully put together and described.

With reference to the second period, dating from the appearance of ataxy, we are told that it occasionally begins with fever. It appears to us questionable whether this symptom does not really belong to some other incidental morbid state, which, by the depressed condition of the general forces, brings into prominence the ataxy which may have previously existed in a very slight degree. Among the various tests by combined movements suitable to the investigation of incipient ataxy, the Author mentions making the patients walk backwards, which is a difficult performance for many even at a very early stage.

Dr. Althaus mentions the various theories propounded to explain the ataxic phenomena. He adopts the "sensorial" hypothesis, without bringing any new arguments in its support. He applies the term "static ataxy" to describe disturbances of equilibrium; whereas Friedreich, who introduced the expression, included under it ataxic movements during apparent rest, quite independently of disturbed equilibration.

Under the heading of the differential diagnosis, diabetes mellitus is especially mentioned, which presents certain nervous symptoms not unlike those of incipient tabes. These, however, we think, can prove perplexing only when the urine is not tested for sugar. It would certainly be difficult in a case where both diseases coexisted to refer each symptom to its true origin; recent French observers have stated that the knee-jerk is absent in typical cases of diabetes. The discussion of the prognosis and therapy of tabes offers no point of special interest. Where syphilis exists, inunction of oleate of mercury is recommended. Ergotin is said to have occasionally good results. Short and weak electrical currents are supposed to be the best. Strychnia is not recommended.

In conclusion, we are bound to acknowledge, much to our regret, "that the darkness which has hitherto," viz. up to the publication of Dr. Althaus's book, "surrounded the question of tabes," has not in the least been dissipated by his exertions. Neither was it required to make us conscious of the imperfection of our know-

ledge, or inspire us with a wish to learn more on a subject which admittedly constitutes an ample field for investigation. Books which are mere compilations never serve to stimulate research.

Prof. SCHULTZE, M.D. (Heidelberg).

On the Functions of the Marginal Convolution. By E. A. SCHÄFER, F.R.S., and VICTOR HORSLEY, B.S., F.R.C.S. Reprint from the 'Proceedings of the Royal Society,' March, 1884.

THE elaborate researches of Professor Ferrier, following the very suggestive but incomplete experiments of Hitzig and Fritsch, clearly showed the fronto-parietal region of the outer convex surface of the cerebral cortex to be separable into distinct areas, which regulated the movements of very different groups of muscles.

The centres thus described and mapped out by Ferrier included those for movements of both limbs and the face and neck. The neck muscles, which were noticed to be represented on the outer surface of the hemisphere, were the rotators of the head.

Schäfer and Horsley have completed this list of motor centres by finding that the trunk muscles are also represented (as might have been imagined) by centres in the cortex cerebri, and that these centres are placed in the marginal convolution. Most experimenters had previously stated that the mesial surface of the cortex of the hemisphere was not excitable, and that at any rate it did not contain motor centres other than some similar to those on the outer surface of the hemisphere, and, further, this statement only rested upon one experiment.

The methods employed by Schäfer and Horsley were those originally employed by Ferrier, viz., stimulation of the cortex by the current obtained from the secondary coil of a Dubois Reymond induction coil, with Helmholtz' side wire, to equalise the effects of the make and break shocks, the current being applied to the brain by means of platinum electrodes, which were carefully insulated to their blunted tips and bent so as to make it possible to introduce them into the longitudinal fissure without hampering in any way the circulation in the cortex. The current employed was almost invariably that obtained from one Daniel cell, and the secondary coil was usually between 7 and 8 cm. from the zero point on the side scale of the primary coil. The current thus obtained gave a distinct but not painful sensation when applied to the tip of the tongue. The animal being anæsthetised with ether (both chloroform and morphia giving inconstant and uncertain anæsthetic effects), the marginal convolution was reached by removing from the vertex a small corresponding oblong piece

of the skull, and turning up towards the middle line a narrow flap of dura mater. The large veins which enter the superior longitudinal sinus from the outer surface of the hemisphere were carefully avoided, and the electrodes gently slipped between the falx and the mesial surface of the hemisphere.

In this way the experiment was performed with extremely little disturbance of the parts, a point of the greatest importance.

The authors discovered that only a portion of the marginal convolution is excitable, and it is very interesting to note that this area exactly corresponds in extent (antero-posteriorly) with the excitable portion of the outer surface of the hemisphere. Thus it is limited anteriorly by a line which passes along the front of the centre for the rotation of the head, or, in other words, which is opposite the function of the posterior and middle thirds of the superior frontal convolution; while, posteriorly, movements can be excited by stimulation as far back as a point opposite the centre of the parietal lobule. The posterior third of the marginal motor region consequently includes the paracentral lobule of Betz, which that observer showed to contain very numerous large pyramidal motor nerve corpuscles.

The rest of the mesial surface of the hemisphere the authors find to be inexcitable. This is of course what might have been expected of the gyrus fornicatus, seeing that there is scarcely any connection between it and the direct pyramidal tract.

The centres for the trunk muscles Schäfer and Horsley find to be arranged in the excitable portion of the marginal convolution, in an order from before back, which corresponds both with the arrangement of the muscles in the body, and also with the related centres for the limb muscles.

Thus arranged in the following order from before backwards, we find the centres for

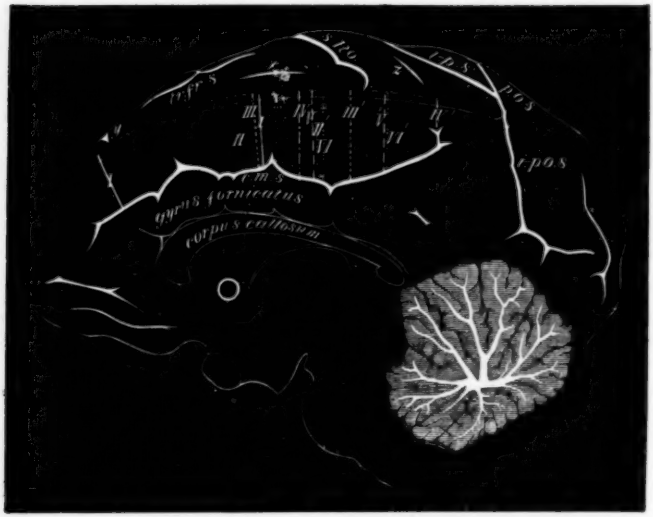
(1.) Flexion and extension of elbow.
(2.) Adduction of shoulder.
(3.) Retraction of shoulder blade often combined with outward rotation of arm.

Owing no doubt to the fact that the trunk muscles are practically the servants merely of the limbs, and can be shown from daily experience rarely to move except to fulfil the requirements of the extremities, the centres for the movements of the trunk in the marginal gyrus are more complex than those centres of the extremities situated on the outer surface of the hemisphere, and therefore when excited by stronger currents, they cause perfectly co-ordinated action of the distal segments of the limbs as well as contractions of the trunk muscles.

(4.) Arching of dorsal region of trunk (spine) with lateral flexion of spine, so that it is arched with the concavity towards the opposite side.
(5.) Same movements in the lumbar region.
(6.) Flexion of hip. Iliopsoas muscle.

(7.) Extension of hip, glutæi, &c.
(8.) Hamstrings and extensors of knee.
(9.) Movements of ankle (flexion, extension, eversion, and inversion, &c.).
(10.) Movements of digits.

It will thus be seen, that, as first stated, we find, even with a moderate stimulation, that groups of muscles are thrown into action which belong both to trunk and periphery. Schäfer and Horsley show, however, that in the middle of each "centre" a minimal stimulus evoked contraction often of a single muscle, and



I to I'. Motor region of shoulder and arm; II to II', of trunk muscles;—III to III', of hip muscles;—IV to IV', of thigh muscles (hamstrings chiefly);—V to V', of ankle;—VI to VI', of digits. Weak excitations in each centre produce localised effects; stronger ones, associated movements of muscles innervated by the overlapped region.

always action of only those muscles which had precisely the same effect on the movements of one joint, or, as in the particular case of the spine, a series of similar joints.

The centre of the thigh muscles is just opposite the upper end of the fissure of Rolando, and consequently occupies the site of the paracentral lobule. The leg centre consequently dips over into the median fissure a good deal more than indicated by Ferrier.

Schäfer and Horsley publish in a note an important experiment as a corollary to the above researches, in which they have removed

not only the centres on the outer surface of the hemisphere, but also their centres for the trunk muscles as well. The result is absolute hemiplegia. The paralysis of all the muscles on the opposite side of the body is apparently complete. This experiment of itself would show that the trunk muscles of one side are for the most part governed by the opposite hemisphere; and the results of stimulation show the same thing, for observation of bilateral movement following unilateral stimulation was very exceptional. Further, they have found, by microscopical examination of the lesion and spinal cord, that there are very marked descending degenerations in cases where they extirpated the excitable portion of the marginal convolution only. The bearing of this upon the explanation why the trunk muscles in hemiplegia so often escape paralysis, or are only temporarily affected, will be obvious, and Mr. Horsley published in the 'Lancet,' of July 5th, 1884, a mode of accounting for this apparent peculiarity in the light of the above-mentioned researches. An abstract of his views will be found at page 132 of the present number.

A. DE WATTEVILLE.

Histoire et Critique des progrès réalisés par la physiologie expérimentale et la méthode anatomo-clinique dans l'étude des fonctions du cerveau. Par le DR. LEVILLAIN.—Paris: Delahaye, 1884.

IN a volume of about 300 pages the Author gives us a very readable—if partial—account of the growth of our knowledge concerning the functions of the brain. As he is apparently unfamiliar with any language but the French, it is only natural that he should fail to give a proportionate share of his attention to contributions from other countries. But it is a serious blot on the book, and one which reduces its scientific value considerably, that the experiments and views of such men as Goltz and Munk are not mentioned, and that even their names do not appear in the otherwise very incomplete bibliography. Several Italian observers also ought to have received some notice at the hands of the Author. England is represented in this account by Hughlings-Jackson and Ferrier. To the former, Dr. Levillain recognises the merit of having been first to admit the existence of motor functions in the cortex. For though as early as 1827 Bravais had described cases of what is now known as cortical or Jacksonian epilepsy, he had failed to interpret the phenomena correctly. And Bright likewise, who had clearly described cases of the same nature, did not succeed in discovering that which alone gives a scientific value to clinical and pathological observation, viz. the true explanation, the law which connects the lesion with the symptom. He seems to have accounted for the existence of convulsions without loss of

consciousness by assuming that the irritation was transferred from the point of the lesion to the subjacent motor ganglia.

As the title of the volume now before us would lead one to anticipate, the Author deals first with the data of experimental physiology. After reviewing the early period (Majendie to Longet and Vulpian, 1820–1870), he divides the more recent experiments under three headings: "Method by Electric Excitations,"—"by Interstitial Injections,"—"by Limited Extirpations." The second of these methods never gave any reliable results, as might well have been expected. The third, on the other hand, since Ferrier and others introduced the antiseptic mode of operation and treatment into it, has yielded brilliant results. But the Author, who, as we have already mentioned, never alludes once to the experiments of Goltz and Munk, does not seem to be aware of the importance of "Listerism" in cerebral surgery, as practised on monkeys. In fact, it is probably owing to the neglect of due precautions in the after-treatment, and to the complications arising therefrom, that the operative procedures of Munk and others give results discrepant from those obtained in this country. Much idle talk has been expended over the first method. The "diffusion" of the electric current through the underlying tissues has been very erroneously brought forward by opponents as sufficient to explain the results obtained from the excitation of a limited area of the cortex. Any one conversant with the physical laws of electricity, and possessing sufficient practical experience of its effects as observed in the application to the organism, will have no difficulty in reducing the objections raised on that score to their just value. This technical difficulty is a purely imaginary one; moreover it is illogical; for if a sufficient diffusion did take place under the conditions of the experiments, it would not support the position assumed by the antilocalisators. It would obviously be as fatal to their position to demonstrate that the efferent fibres from certain regions of the cortex are motor, as to show that the cortical cells of those regions are electrically excitable.

The second part of Dr. Levillain's book gives a *résumé* of the labours of Charcot and his school, from the anatomo-clinical point of view. English and German contributions to the subject, including Nothnagel's classical *Topische Diagnostik*, receive but scanty recognition in the shape of a casual reference. But as it stands, this part forms an interesting chapter in the history of the question. The subject of Aphasia is touched upon, but inadequately. Those of our readers interested in it will, however, find it more exhaustively handled in the recent thesis of another pupil of the Salpêtrière, Dr. Bernard. A revolution has at last been successfully accomplished in the doctrines which have kept French observers spell-bound, as it were, since the days of Broca and Trousseau. Prof. Charcot has taken up the question, and with his characteristic force and clearness of exposition introduced to his disciples, and to the profession generally, the results reached by the psychologists of the new English school, which so admirably fit in with the recent pathology and physiology of the brain as an organ of

mind. The labours of H.-Jackson, Bastian, Broadbent, as well as Wernicke and Kussmaul and others on Aphasia, have now at last been recognised in France, where, with few exceptions, such as the thesis by Skwortzoff, the literature of the subject had remained confined within the narrow original limits.

An interesting case is described, at page 177, of sudden loss of the power of reviving the visual impression of objects (without verbal blindness). A gentleman of high intellectual culture had his visual memory developed to such a degree as to rely upon it for the greater part of his psychical operations. One morning he awoke with a feeling of confusion, and discovered that he had lost the power of revivifying past visual impressions, or recognising objects and persons he knew before. This case, as the Author remarks, is a good illustration of the distinct localisation of the various memories which form the basis of the manifestations of mind. It is from observations of this order, patiently collected and confirmed by autopsies, that our future knowledge of the higher functions of the brain has to be elicited. Not that the resources of experimentation are exhausted, or that its assistance can be dispensed with; for even where clinical investigation must, from the nature of the problems, lead the way, the discipline of comparative and physiological methods is required to act as a wholesome check upon hasty generalisations.

A. DE W.

The Brain and its Functions. By I. LUYS, M.D. Second Edition. London: Kegan Paul. pp. xviii., 327.

The Brain as an Organ of Mind. By CHARLTON BASTIAN, M.D., F.R.S. Third edition. London: Kegan Paul, 1885. pp. xviii., 708.

THE volumes before us both belong to the International series, but beyond this fact, and the similarity of their titles, have but little in common.

Of Dr. Bastian's work we need not say much here, for we trust that it is already in the hands of every reader of 'BRAIN.' Though not immediately intended for professed physiologists or medical practitioners, it constitutes a valuable contribution to the literature of the subject, and we are glad to see new editions of it rapidly succeed to one another.

We regret not to be able to say the same of Dr. Luys' book, which in our opinion is calculated to mislead and hopelessly confuse the unwary reader. The first part is anatomical, and contains an account of the author's peculiar views concerning the structure of the brain. Dr. Luys starts from the extraordinary statement, that microphotographs of brain sections afford clearer views of the structural details than the direct examination of them. As an instance of the results he obtains by his methods, we will take the optic thalamus, in which he thinks he has discovered four

sensory ganglionic centres, wherein olfactory, visual, sensitive, and auditory impressions are respectively "condensed" in their way up to the cortex. The sense of taste, it will be observed, is not represented in this arrangement, which, after the researches of Forel and others, we may well characterise with Schwalbe ('Lehrbuch der Neurologie,' p. 713) as fantastic and hopelessly erroneous.

It is difficult to understand how observers can still be found who, closing their eyes to the clearest teachings of physiology and pathology, pretend to solve the problems of cerebral function by purely anatomical data. Quite recently attempts were thus made to show that the decussation of the descending tracts took place in the corpus callosum only; and again that Broca's convolution is not a motor centre of speech, because fibres cannot be traced from it to the internal capsule. The whole of Dr. Luys' doctrine is tainted with similar tendencies. It is enough for him to find that the cerebral cortex contains layers of small and of large cells to argue that it has sensory and motor functions throughout its extent, because in the cord the motor cells are large, the sensory small. The calm assurance with which the author describes his theories is never once troubled by the consciousness, that he is in flagrant opposition to some of the best established results of modern science. He never alludes to them, nor lets his reader suspect that his opinions are shared by no other observer of note.

The second and third parts are devoted to the "general properties of the nervous elements," and to "the evolution of the processes of cerebral activity." We shall not attempt to criticise Dr. Luys' treatment of this part of the subject, but content ourselves to illustrate it by means of a quotation or two, typical of the style adopted by the author. "This form of sensibility [moral] which runs riot in spite of us, these plexuses of the *sensorium commune*, which comprehend in themselves all the diffuse sensibilities of the organism, represent, then, a sphere of nervous activity in erethism, always living, always feeling, in the bosom of which our total personality lives and vibrates. There, in this mysterious dwelling, it is in perpetual intercourse with the perpetual movement of the operations of cerebral life." "The human personality is seized upon by the arrival of the excitation arriving from the personal world. It enters into contact, and becomes associated with this; and from this intricate connection results a true intra-cerebral automatic radiation, which produces the apparition of a series of agglomerated secondary ideas. But the matter does not stop there; this inner personality having been thus seized upon, its sensibility having been touched in any manner whatever, has reacted by virtue of the vital forces that vibrate in it in a latent condition,—it has been affected in the direction of its most profound affinities, and necessarily this reactionary period betrays itself by an unconscious desire for such or such a definite object, and by a repulsion from such or such another."

Such are the "functions of the Brain," according to Dr. Luys.

A. DE W.

Les Troubles de la Parole. Par le Professeur A. KUSSMAUL. Traduction française par le Dr. A. RUEFF. (pp. 375.) Paris: J. B. BAILLIÈRE, 1884.

THE classical treatise on the diseases of Speech by Professor Kussmaul first appeared as a volume in Ziemssen's 'Cyclopædia.' English readers who do not know German and do not have access to the American translation of the series, have hitherto been debarred from the perusal of the work before us, the only one of its kind. It will therefore be welcome news to many that Kussmaul's monograph has been placed within their reach by the careful translation into French which Dr. Rueff has lately supplied. We need not go into any details concerning the method adopted by the Author in the treatment of the difficult subject of Language. His teaching has been generally recognised as sound as well as exhaustive; and his views have been generally adopted by German writers. Those of his readers, however, who are conversant with the results of this English school of psychology will notice the disadvantage at which the Author stands in the introductory chapters, from his inability to shake off the semi-metaphysical standpoint still current among many of his countrymen. The discussion on aphasia, and of articulatory disturbances, which forms the bulk of the work, is full of facts and rich in suggestive thoughts.

A. DE W.

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1. *Rivista di Filosofia Scientifica.* Diretta da E. MORSELLI.
2. *L' Origine dei fenomeni psichici.* Di G. SERGI. Fratelli Dumolard, Editori. Milan and Turin, 1885.

THE political resurrection of Italy has been speedily followed by a scientific renaissance among its people. On every side signs of increasing activity become manifest. We have now before us some numbers of a new 'Review of Scientific Philosophy,' which is just entering upon its fourth year of existence, and which is conducted on much the same lines as the well-known 'Revue Philosophique,' edited by M. Ribot. Positive psychology and neurology are amply represented in its pages. Thus we may notice an interesting "Study of Experimental Psychology: on the Time of Perception of Colours," by Buccola and Bordoni; articles on "The Notion of the Individual in Biology," by Bonelli; and "Researches on the Influence of Hypnotics on the Duration of Psychical Acts," by Cervello and Coppola. Professor Fano gives us a discourse on "Physiology as an Autonomous Science;" and finally, Sergi publishes a chapter, "On Psychical Acts as Functions of the Organism," from his new work since published.—'L' Origine dei Fenomeni Psichici e loro significazione biologica,' is a title

sufficiently descriptive of the tendency of the book, which takes its standpoint on the modern monistic hypothesis. It is pervaded with the true Darwinian and Spencerian spirit, and the author fearlessly deduces his conclusions, driving them to their uttermost consequences. Consciousness arises and is developed in the series of animal life, as "a function of protection." With a great fulness of detail, the Author unfolds to the reader the ascending steps by which the evolution of mind is accomplished according to the principles of the struggle for life.

This new work by the Professor of Anthropology in the University of Rome forms a volume of the "International Series," and will therefore, very probably, become accessible to the bulk of English readers at no distant date.

A. DE W.

Zur Einleitung in die Elektrotherapie. Von Dr. C. W. MÜLLER. J. F. Bergmann, 1885. 1 vol. 8vo. (pp. 187).

THIS little book embodies the results of most praiseworthy efforts in the scientific direction fortunately taken by Electrotherapeutics of late years. The Author takes the same standpoint which we ourselves have always assumed, viz. that the fundamental question in electric treatment is one of physics, and not of physiology. It is with much satisfaction that we see the question of current measurement occupy a more and more prominent position in the various papers and books published on this subject, and the unit we first proposed (milliweber, or milliampere) adopted by all good observers. The work now before us consists of two parts. In the first the Author furnishes us some very precise data concerning the various galvanometers intended for medical use; he comes to the conclusion that the new astatic vertical instrument of Hirschmann is the best. The second part is much more practical, and deals with the question of the density of the current in actual applications to the human bodies. By density is meant the ratio between the strength of the current, and the surface to which it is applied. This surface is obviously equal to that of the electrode. Hence the expressions $\frac{1}{5}$, $\frac{1}{10}$, $\frac{2}{15}$, $\frac{2}{24}$, and the like, indicate that current of 1 or 2 milliamperes is applied by means of an electrode of 5, 10, 15, 24 square millimetres. The time during which the current is allowed to flow is also indicated, and thus a very accurate system of dosage is obtained. The Author's experience leads him to formulate the aphorism that electric applications should be weak, short, and frequent. Indeed, in his practice he reminds one of the homœopath and infinitesimal doses. We must confess that we have never tried such a plan, for the simple reason that we have found a more decided course of action generally useful, especially in cases of organic disease. To give an instance; instead of a current of $\frac{1}{18}$ for one minute daily, as recommended by Dr. Müller in sciatica, we have often found great and almost immediate relief from

applications of currents as strong as the patient can bear, with very large electrodes ($\frac{?}{150}$ or more), for 10 to 20 minutes, with a moderate faradic current flowing at the same time. In all except very chronic cases this strong application is to be repeated more than once a day.

We must, however, recommend Dr. Müller's little book as a thoroughly exact, honest, and painstaking performance. He writes obviously with enthusiasm, and for the instruction of the reader rather than for his own interest or glorification. He is at the same time scientific and practical, and the spirit he displays stands in refreshing contrast with the tendencies displayed by too many writers on electrotherapeutics.

A. DE W.

A Practical Treatise on Disease in Children. By EUSTACE SMITH, M.D. London: Churchill, 1884. 8vo. pp. 840.

THIS volume is certain to become popular among medical practitioners, for whom it is intended from the essentially practical standpoint assumed throughout by the Author. On every page we discover evidences of its origin; not the outcome of painstaking compilation of authorities, but the natural flow from a mind well stocked with personal experiences. Its function is to serve as a guide at the bedside, not as a didactic teacher at the reading-desk; and so the subjects of diagnosis and treatment have received the greatest share of the Author's attention.

Dr. Smith did well to alter the customary "Diseases of Children," into "Disease in Children." There are no diseases which deserve the former name; but, on the other hand, the physiological characteristics of childhood which modify disease in many ways, and the peculiar difficulties of diagnosis and special requirements of treatment in the case of patients in the first period of life, fully justify the distinction implied in the latter.

This remark applies more particularly to nervous disorders, to which 140 out of a total of 800 pages are devoted in the work before us. The nerve-centres form the most differentiated structures of the body and are the last to reach their full differentiation. The new-born child is an automaton, and his psychical existence a mere manifestation of vegetative reflexes; under the influence of the various external stimuli, the rapidly growing hemispheres are the seat of extraordinarily active processes, and the motor tract is subject to ceaseless demands from the higher centres. Inhibition is still in abeyance, and there is no localisation of single excitations, which, diffusing themselves over the whole cortical surface, give rise to disproportional external effect. It is this diffusibility of nervous processes which led psychologists to speak of the "spontaneity" of the nerve-centres, and gives a peculiar aspect to many of the morbid phenomena of childhood.

The immature condition of the higher centres, and the greater

activities of the vegetative functions in early life, would have led us to expect *à priori* that the treatment should differ from that adopted for the adult, not only quantitatively, but qualitatively. Nothing can be more fallacious than the mathematical dosage of remedies adopted in the usual textbook of Materia Medica, when no account is held of any circumstance except of age. Dr. Smith has fortunately quite emancipated himself from the orthodox superstition; and we are glad to see him recommend, for instance, arsenic in 10-drop doses or more in chorea, where our experience agrees with his as to the value of treatment, if heroic enough. Tincture of belladonna, again, may be given beneficially in doses of thirty drops to infants, upon whose immature brains the drug cannot have any toxic effect, whilst such quantities exert a distinct influence upon the lower reflex processes. The author's directions as to the hygienic and dietetic management of children are full and practical; and the whole question of treatment is handled with a mastery which only years of thoughtful experience and observation can impart.

A. DE W.

Lectures on Mental Disease. By W. H. O. SANKEY, M.D.—2nd Ed.—London: Lewis, 1884.

WE welcome with pleasure the second edition of Dr. Sankey's 'Lectures on Mental Diseases.' Few men in this country have had a wider or more varied experience of insanity than Dr. Sankey, and no one perhaps has a greater right to speak with authority on the subject than himself. Excepting the first 99 pages, devoted to mental science or physiology, the book is eminently practical. The first part, notwithstanding the very excellent plates of microscopical structures which illustrate it, might, we are inclined to think, have been omitted, or at any rate much curtailed, with advantage. The remainder and by far the larger part of the book is all good. Dr. Sankey prefers to include all forms of insanity, excepting General Paresis and Idiocy, under the head of Ordinary Insanity, on the ground that they are all phases or stages of the same disease. In this view he is probably right. Nevertheless, under this head he describes the generally recognised varieties of the disease just as most other writers do. And considering that writers generally would agree with him in opinion, and that their separate groups are made, as are his separate descriptions, mainly for convenience of arrangement, the difference between him and them seems apparent rather than real. At any rate it is not very important. We do not say this with the object of fault-finding; and indeed are disposed to admit that there may be some advantage in impressing upon students the fact of the essential identity of most varieties of insanity. The chapters on treatment, and on the legal relations of insanity, are full and satisfactory.

J. S. BRISTOWE, M.D.

An Introduction to the Study of the Diseases of the Nervous System. By THOMAS GRAINGER STEWART, M.D., F.R.C.P.E., &c. Edinburgh; Bell and Bradfute, 1884.
Diseases of the Spinal Cord. By BYROM BRAMWELL, M.D.—Second Edition. Edinburgh: Pentland, 1884.

THE clinical study of nerve disease and disorder has come to be more and more arduous with advancing knowledge; and it is doubtless true that some guide is requisite for those who are beginning to investigate such cases, and to search for causes among the multifarious abnormalities which come under their observation. There is certainly room for such a book as that before us, which tells the student what to look for in cases of supposed nervous disease, and how to look for it. It is well, especially in this department of medicine, that a clear idea should be gained of what the symptoms are which point to nerve disease, and what the known or probable causes of such symptoms may be, before the so-called "diseases" of an established nosology are each of them separately studied. In such a book as this, originality is of course not to be looked for, nor would it be, indeed, desirable; and it is equally impossible in the space of the volume for acknowledgment to be made of the various thoughts and illustrations taken from other authors. It is in the main a compilation. But Professor Stewart has done well the work he has undertaken, especially in the part relating to symptomatology. The first three lectures on the medical anatomy of the nervous system would be of greater value as lectures, clearly illustrated, than they are for perusal by the reader; one at least of the diagrams is unintelligible in its minuteness. The altered symptoms, however, occurring in disorder of the various departments of the nervous system are very clearly set forth, and the description is frequently accompanied by excellent plates. The book, as a whole, is carefully written; and will doubtless be widely used by those to whom ampler volumes are inaccessible.

Dr. Bramwell's book well deserves the success it has already met with. Translations into German, French and Russian, and a second English edition reached within two years and a half of its first publication, show how keenly it has been appreciated by the students and practitioners for whom it is intended. The characteristic feature of the work is the fulness and perfection attained in the illustrations. In this edition the number of the beautiful chromolithographs and drawings representing the normal and pathological structure of the cord has been still further enlarged; and a chapter added on "Concussion;" whilst the text bears the evidence of careful revision throughout.

H. B. DONKIN, M.D.

Critical Digest.

ON THE COMBINATION OF LATERAL AND POSTERIOR SCLEROSIS IN THE SPINAL CORD.

BY J. A. ORMEROD, M.D.

In the classification of chronic degenerative diseases of the cord, a distinction is generally admitted between those that are "systematic" and those that are "diffuse;" the systematic diseases being those that affect only certain definite physiological tracts, and spread only in the direction of the affected tracts, the diffuse being indiscriminate in their onset and mode of extension. The division of the cord into such physiological tracts has been based on the following methods: (1) observing the march of degeneration in fibres separated from their nutritive centres, [secondary degeneration: method of Türck and Waller], (2) observing the different times at which different sets of fibres reach their full development [method of Flechsig]. A striking agreement obtains in the results of these two methods; which is rendered still more striking by the fact, that (3) certain chronic diseases of the cord whose precise causation is unknown (primary degenerations) map out, at least in their simpler forms, corresponding regions of the cord. These are the "systematic" diseases, or as they may be more shortly called, tract-diseases.

The first and second of these methods have been principally applied to the white substance; and we shall in this article consider disease of the white substance only, though certainly the selective power of disease is manifested also in the gray. But under our heading will be included at least two varieties of disease —one in which the sclerosis of each column coincides with the known physiological tracts contained therein (for this we propose the name multiple tract disease); the other, in which the sclerosis of one or other column (generally it would appear the lateral column) does not coincide with known tracts, though too regular in its distribution to be called in the strictest sense diffuse.[1]

We shall first give a brief abstract of such cases as we have been able to find in foreign literature.[2]

[1] It may be noticed that Westphal ('Arch. für Psychiatrie,' etc., vol. viii.) gives a case of posterior sclerosis combined with disseminated sclerosis of other parts.

[2] We have unfortunately been unable to obtain the account of a case by Mr. L. Hamilton, 'New York Medical Record,' 1879.

(1.) PIERRET. 'Arch. de Physiol.,' vol. iv., 1871–1872:—

A female, æt. 60. Complete paralysis of the lower limbs for twelve years; which had been preceded by weakness and pain in them. Sensibility absent in the legs: arms thin, and their movements inco-ordinate. State of torpor.

Post-mortem.—Degeneration of the whole of the *posterior* columns and atrophy of the posterior nerve-roots. *Lateral* columns, posterior parts diseased, chiefly in the lumbar region.

[This may have been simply an example of the transverse spreading of the sclerosis from the posterior columns to the lateral. It is, however, quoted by Westphal in his article, to which we shall presently refer.]

(2.) PREVOST. 'Arch. de Physiol.,' series ii. vol. iv. 1877.

Male, æt. 51. Duration of disease, between seven and ten years. Initial symptoms, pains in loins, weakness in legs and in right arm, irritation in skin of right arm, difficulties in defæcation. Then increase of weakness till patient could not walk or sit up, double vision, progressive amaurosis. While under observation the lower limbs were almost paralysed, the upper limbs ataxic, sensibility diminished. Reflex movements (to pinching, tickling, &c.,) were very violent, especially on the right side. Finally, emaciation set in: death from bedsore.

Post-mortem.—There was in the *posterior* columns sclerosis, of an extensive area, throughout the whole length of the cord, but most extensive in the dorsal region. In the *lateral* columns, sclerosis, also most marked in the dorsal region, and diminishing gradually as the lumbar and cervical regions were approached. It involved chiefly the posterior part of the lateral columns, but extended forwards along their periphery. In the dorsal region, it dipped deeply into the substance of the cord. Except in a few sections the lateral sclerosis was not continuous with that of the posterior columns, and the author insists that it could not be regarded as a simple extension of the latter.

The violence of the reflex movements (the skin-reflexes only are alluded to) led him to conjecture, during life, the existence either of a transverse myelitis or a lesion of the lateral columns.

(3.) KAHLER AND PICK. 'Archiv für Psych.,' vol. viii. 1878.

A case clinically resembling the hereditary ataxia of Friedreich, remarkable post-mortem for the exactness with which the posterior columns, cerebellar tracts, and pyramidal tracts were picked out by disease. Of this case we have already given an abstract in BRAIN, No. xxv., p. 114.

WESTPHAL. 'Archiv für Psychiatrie,' &c., vols. viii. and ix., 1878–1879, gives a series of five cases:—

(4.) Female, æt. 45. Duration, nine years in all. Disease began with pains in back and in left leg, then numbness in fingers, shooting pains in upper and lower limbs, double vision. In five years' time difficulties of micturition, and of defæcation, and of

walking began. While under observation (a period of three years) there was noted—paralysis and impairment of sensibility in the lower limbs, ataxy and impairment of sensibility in the upper limbs; paralysis of the bladder, paralysis in the districts of the right third and sixth nerves, and of both trigeminal nerves; affection of the cornea, amblyopia, ataxy of the facial muscles, attacks of dyspnœa and delirium: general emaciation, especially in the legs.

Post-mortem.—The following nervous lesions were found:—

Chronic lepto-meningitis: degeneration of the posterior nerve-roots. Within the cord the *posterior* columns were sclerosed, over nearly their whole area, through the whole length of the cord. There was also degeneration of the *lateral* columns (differing somewhat in its microscopical characters from that of the posterior) which occupied, in the lumbar and dorsal regions, an area at the posterior part of the columns not reaching to the periphery of the cord (crossed pyramidal tracts?). In the cervical region this area was healthy, but here at the periphery, in the middle part of the columns, there was a thin zone of disease, faintly marked and only to be made out with the microscope.

Further there was degeneration of the ascending root of the trigeminus, reaching upwards to the decussation of the pyramids: atrophy of the trigemini and partial atrophy of both optic nerves.

(5.) Female, æt. 51. Nineteen years before her admission she had noticed, during pregnancy, a numbness of the abdomen; next her gait became waddling. After an abortion, weakness of the right leg appeared: then a dislocation of the hip joint in consequence of joint-disease. [The author does not consider the joint disease to be of the special form described by Charcot.] Five years before admission there appeared numbness and weakness of the upper limbs, and four months before admission inability to walk, difficulty of micturition and defæcation, double vision, impairment of speech, asthmatic attacks. After admission, there was observed complete paralysis of the lower limbs, paresis of the upper limbs, impairment of sensibility in both upper and lower: no rigidity. There were nervous disturbances in the districts of both trigemini, also increased flow of saliva, congestion of the face, sweats, and vomiting. Later still, impairment of speech and of swallowing, general emaciation, death.

Post-mortem.—There was chronic lepto-meningitis; and in the cord, sclerosis universal or nearly so of the *posterior* columns. In the rest of the cord there was disease reaching from the anterior fissure to the tip of the posterior cornu, involving an area which varied at different levels, as follows: in the cervical region there was only a thin zone of disease reaching along the periphery and along the anterior border of the posterior cornua; but in the dorsal and lumbar regions this zone became broader, and extended more and more deeply into the substance of the lateral columns. There was also some disease of the gray substance of the anterior cornu, most marked in the lumbar region.

(6.) Female, æt. 45. Duration of disease, six years. It began with bouts of pain at the left lower ribs, and vomiting; next there developed gradually weakness and anomalies of sensation in the legs and in one or two fingers; awkwardness in using the hands, transitory diplopia, progressive amaurosis, numbness of the left face, and left side of buccal cavity. While under observation (a period of two years), the weakness gradually increased, first in the legs, then in the arms (the left especially); the gait, at first somewhat ataxic, became paretic in character; the movements of the legs, examined while the patient was lying down, were ataxic, but finally evinced much loss of voluntary power. The legs, however, were subject to involuntary movements. In the left arm there was ataxy and loss of power; in the right, slight ataxy. The impairment of sensibility in the upper and lower limbs gradually increased and spread to the abdomen. Girdle-sensation present; skin reflexes normal; knee and foot phenomena absent; no paralysis of bladder; impairment of sensibility in the district of both trigemini; flushings of the face and neck; sweating; frequent pulse; painful vomiting; much general emaciation.

Post-mortem.—There was pulmonary and intestinal phthisis; and in connection with the nervous centres, chronic pachymeningitis of the convexity of the brain; adhesions of the spinal dura and pia-mater, especially on the posterior aspect of the cord. Within the cord, sclerosis tolerably universal of the *posterior* columns, and also of a zone at the periphery of the *lateral* columns, but this was nowhere so broad as in the last case. There was also atrophy of the ascending roots of the trigemini, of the trigeminal, optic, and olfactory (?) nerves.

(7.) Male, æt. 45. Duration of disease, four or five years. First, stiffness in the legs, girdle-sensation, transient pains, of a tearing character, in the legs; then, after an injury to the knee-joint, numbness and pricking in the feet, difficulty in walking, slight troubles of micturition and defæcation. While under observation, there was paresis of the lower limbs, stiffness of the joints, spastic gait, foot-clonus; sensory troubles in the upper limbs. The patient had chronic lung disease; diabetes mellitus supervened, an abscess in the rectum formed, and death followed.

Post-mortem.—Fluid was found in the sac of the dura-mater; adhesion between the dura and pia. There was disease both of the lateral and posterior columns, the distribution of it being much as follows. Its area was most extensive in the upper dorsal region; the *posterior* degeneration here affected both the columns of Goll and of Burdach, higher up the columns of Goll only, lower down it disappeared altogether. The *lateral* degeneration in the dorsal region extended inwards from the periphery over a considerable area of the posterior and middle part of the lateral columns; above the dorsal region, only over a thin zone at their periphery, while below the dorsal region it became more and more limited to the area of the crossed pyramidal tracts.

Thus the distribution of the disease above and below the dorsal

region corresponded to that of a secondary degeneration proceeding from a transverse lesion of the middle part of the cord (columns of Goll and cerebellar tracts above, pyramidal tracts below), and this explanation of the facts is advanced by some recent writers.[1] Westphal himself thinks that, in the absence of a distinct patch of transverse myelitis, the disease must be considered to be primary; it corresponds to what he has observed in paralytic dementia. Clinically the spastic gait and exaggeration of tendon reflexes were so marked, that had it not been for the slight sensory symptoms and the disturbances of micturition, the case might have been taken for pure and simple lateral sclerosis.

(8.) Male, æt. 44. Duration of disease, six years; death from peritonitis, caused by ulceration round a gall-stone.

Pain in the back, transient numbness of thorax, transient diplopia and weakness of the bladder, diminished sensibility in left upper limb. Three years later, difficulty of walking and sense of constriction at the thorax. After admission, increased disturbances of sensibility, impairment of muscular sense, retarded sense of pain. Ataxy of all four limbs, and involuntary movements of them. Double amaurosis, nystagmus. No rigidity, absence of tendon reflexes.

Post-mortem.—Chronic spinal lepto-meningitis; of the *posterior* columns, tolerably universal degeneration; of the *lateral*, degeneration which in the cervical and dorsal regions was asymmetrical, involving on the left side the middle part of the column near its periphery, on the right side its posterior part and more deeply. In the lumbar region and below, the lateral degeneration became more symmetrical, and its area corresponded more nearly with that of the crossed pyramidal tracts.

Westphal's remarks upon these cases are important; and though we shall have occasion to recur to them later, we may give the gist of them here. The posterior sclerosis was undoubtedly systematic, involving as it did known physiological tracts; but the disease of the lateral columns (in some parts at any rate) corresponded neither to the area of the pyramidal tracts, nor of the cerebellar tracts, nor to a combination of the two. The relation of the disease in the lateral and posterior columns respectively is difficult to determine. Westphal's view, if we understand him rightly, is that both are primary and independent of each other, and that the irregularity in the distribution of the lateral degeneration is more apparent than real, for fibres in different tracts may, he thinks, derive their nutrition from an identical source, and thus fall victims to one and the same primary degeneration. He observed in several of the cases a difference between the histology of the lesions in the two parts of the cord; but he regards the process as essentially the same in both, the posterior sclerosis being however of older date than the lateral. Clinically the diagnosis has to be made between simple disease of the posterior columns and the

[1] MM. Ballet et Minor, 'Arch. de Neurologie,' No. 19.

combined disease; and the chief point of distinction is that, in the latter, symptoms of motor weakness are added to the ataxy. The gait becomes paretic, there is dragging rather than jactitation of the legs, and at a later stage, when the patient is examined lying down, feebleness of movement can be recognised as well as incoordination, though the identification of these phenomena may doubtless be difficult. At any rate, the loss of power actually observed by him is explained most naturally by reference to the lesion of the lateral columns (though the explanation has its difficulties).

Rigidity and spastic phenomena were absent, except in his fourth case (No. 7), where the lumbar part of the posterior columns was normal. Hence Westphal comes to the conclusion, "that in combined disease of the posterior and lateral columns rigidity and spastic contractures do not appear, if the disease of the posterior columns reaches down to the lumbar region and involves that part of the posterior columns known as the posterior root-zone."

(9.) Rabasiu. Virchow's 'Archiv,' vol. 76 (1879), p. 74. Male, æt. 44. Duration of disease, three years. It began with a feeling of weakness and heaviness in the legs, difficulty in walking, stiffness in the legs. Then pains in the lower limbs and genitals; for which, two years after the onset of the disease, the patient took to bed, where he remained for the rest of his life. Double optic atrophy began about six months before his death. The lower limbs were observed to be rigid, the feet in the position of equinovarus. There was occasional clonic twitching of the legs. Yet the tendon-reflexes were but slightly exaggerated. There was slight diminution of sensibility in the feet. Paralysis of bladder and vesical sphincter. Death from bedsore and gangrene of bladder.

Post-mortem.—The spinal meninges were thickened and pigmented. The spinal degeneration was most marked in the dorsal region. Here in the *lateral* columns it reached from the posterior nearly to the anterior cornua and round the periphery of the anterior columns to the anterior fissure. Above and below the dorsal region the disease of the lateral columns diminished in extent. In the *posterior* columns there was degeneration, through the whole length of the cord, of Goll's tracts, and of the peripheral part of the cord just external to Goll's tracts. The histological appearances resembled those of insular sclerosis, viz., thickening of the interstitial tissue, increase of neuroglia nuclei, presence of amyloid bodies, hypertrophy of axis cylinders. The cells of the anterior cornua and of Clarke's columns were normal.

The intensity of the disease was much greater in the lateral than in the posterior columns, and with this fact the author connects the spastic symptoms observed during life. From the combination of these spastic symptoms with the sensory symptoms, bladder trouble, and optic atrophy, the diagnosis of combined lateral and posterior sclerosis had been made.

(10.) Kahler and Pick, 'Archiv für Psychiatrie,' vol. x. 1880,

give an account of a post-mortem (without clinical history), in which they found *posterior sclerosis* limited to Goll's tracts in the upper part of the cord, and involving the posterior radicular zones lower down; *lateral sclerosis* involving the whole length of the pyramidal tracts, and also the periphery of the lateral and anterior columns. This peripheral sclerosis was, in their opinion, partly due to systematic disease of the cerebellar tracts; for there was a paucity of cells in the columns of Clarke; the cells which constitute, as they think, the nutritive centres for these tracts.

Schultze, 'Virchow's Archiv,' vol. 79, 1880, gives two cases.

(11.) The first constituted, during life, one of the cases upon which Friedreich founded his original description of hereditary ataxia. An abstract of the case will be found in 'Brain,' No. xxv. pp. 115–120.

(12.) In the second there was no hereditary history. The patient was a middle-aged man, whose complaint had lasted ten years at the time of his death. First symptom, tearing pain in left leg; in two years' time, ataxy of legs; soon afterwards, optic atrophy, which caused complete blindness in two years more. On admission, paralysis of lower limbs with rigidity (flexion of knees and thighs, with adduction of thighs); absence of patellar tendon-reflex and of ankle-clonus; loss of sensation, except for heat, in the lower limbs. Upper limbs normal. Frequent pains in legs and back. Incontinence of fæces.

Examination of the cord shewed "fibrillar degeneration" of the *posterior* columns in their whole length; and also of the posterior part of the lateral columns. The disease in the *lateral* columns involved not only the pyramidal tracts, but also a zone at the periphery of the middle part of the columns. This peripheral zone corresponded to the area of the cerebellar tract in the cervical region, but in the dorsal region spread beyond it.

There was slight thickening of the pia-mater. The posterior roots were thinned. The grey-matter of the posterior cornua was slightly diseased. The cells of Clarke's columns were atrophied.

Apropos of this particular class of case, Schultze makes the apposite remark, that the disease is too regular in distribution for insular sclerosis, too irregular for genuine tract-disease.

Three remarkable cases are given by Strumpell, 'Archiv für Psychiatrie,' vol. xi. 1881.

(13.) The symptoms resembled those of amyotrophic lateral sclerosis.

Female, æt. 32. Duration of disease, three to four years. It began, after a fall on the back of the head, with feverishness, pain in the head, stiffness of the neck, slight weakness in the right leg. Three months later, decided weakness in the legs; later, slight twitching of them, slight paræsthesiæ, some difficulty of micturition. She was admitted to Hospital a year before her death; there was then considerable paralysis of the legs, with

stiffness and increased tendon reflexes; associated movements of the two legs. Sensibility normal. Slight involuntary (choreic?) movements of the arms. Some inequality of pupils. For a time there was slight improvement under galvanism, but she finally got worse. Wasting of some muscles in the legs and hands was noted; finally, incontinence of urine appeared, and death followed.

Post-mortem.—Strumpell found disease of the pyramidal tracts, and of some sets of fibres in the posterior columns, with slight disease of the cerebellar tracts. The anterior cornua were normal, but the cells of Clarke's columns were atrophied.

There was no meningeal affection.

The affection of the *pyramidal tracts* could be characterised as strictly systematic; both because the area of disease corresponded exactly with that of the crossed pyramidal tracts, and because one direct pyramidal tract was also affected. But it was not secondary to any intracranial lesion, for the pyramids of the medulla and the crura cerebri were normal. It was therefore primary, and propagated, the author thinks, from below upwards.

With respect to the *posterior columns*, although the diseased areas did not correspond exactly with what we know of the physiological tracts in this part of the cord, yet from its bilateral symmetry, and from the length of cord over which the posterior sclerosis extended, Strumpell thinks that it must be classed as systematic, as the lateral sclerosis undoubtedly was.

Roughly speaking, Goll's columns were diseased from the upper lumbar to the cervical region. Besides this, there was in the dorsal region a triangular area of degeneration, with its base at the periphery of the cord, between Goll's columns and the posterior root zones. In the upper lumbar region the posterior root zones, and in the lower lumbar region the whole posterior columns were free from disease. Thus the spastic symptoms and excess of tendon-reflexes can be accounted for in accordance with Westphal's dictum. (The spastic symptoms consisted in intermittent painful contractions rather than in permanent rigidity.)

In the wasted muscle there was no distinct reaction of degeneration during life, nor was degenerative atrophy found post-mortem: neither were the anterior cornual cells diseased. Hence the resemblance to amyotrophic lateral sclerosis was more apparent than real.

(14.) Paralysis and contraction of lower limbs, with impairment of sensibility. Exaggeration of tendon-reflexes: incontinence of urine. The patient was an old and pauper woman; the disease lasted about nine months.

The posterior columns were normal in the lower lumbar region; in the upper lumbar region traces of disease began to show themselves in Goll's columns; further up, the disease became more marked in Goll's columns. In the cervical region certain other portions of the remainder of the posterior columns were also degenerated. In the lateral columns there was well-marked degeneration of the cerebellar tracts, degeneration also of the

crossed pyramidal tracts, less intense than in the cerebellar, but becoming more marked in the lower part of the cord. The gray substance was normal, with the exception of Clarke's columns.

There was, therefore, in these two cases, simultaneous degeneration of those three tracts in the cord which contain long fibres, viz., Goll's tracts, the cerebellar tracts, and the pyramidal tracts. It is remarkable that the degeneration of the pyramidal tract was (in the author's opinion) ascending, while that of the posterior columns was more marked above than below. The areas of disease in the posterior columns were somewhat peculiar: but for details of them we must refer to the original paper.

(15.) Typical tabes, with subsequent paralysis of the lower and paresis of the upper limbs.

Male, æt. 50. Duration of disease, 19 years. First, tearing pain in the legs, and sweating of the feet: in two years' time, difficulty of walking. Blindness for 14 years, deafness for four years. On admission, there was complete paraplegia, the tendon-reflexes were absent, the muscles flaccid, their electro-contractility diminished. There was emaciation, but no fibrillar twitching of the muscles. The patient was absolutely blind, absolutely deaf, and anæsthetic over the greater part of his limbs and trunk. He had to be questioned by tracing out letters on his forehead. Incontinence of urine, cystitis, and a bedsore ended his life.

Post-mortem.—There was almost complete degeneration of the *posterior* columns and posterior nerve-roots, of the *cerebellar tracts* and (in the lower dorsal and lumbar regions) of the *crossed pyramidal tracts.* Also chronic meningitis, and atrophy of the optic and acoustic nerves.

(16.) Sioli. 'Archiv für Psychiatrie,' vol. xi. 1881.

In this case the gray matter was more extensively diseased than in any we have yet considered: there was, moreover, dementia.

Male, æt. 39. Severe fall, followed by coma and transitory paralysis. Slight weakness of the legs remained, which in five years' time began to increase steadily; sensory affections of the legs also began. On admission, there was typical chronic dementia; almost complete paralysis, with wasting of the legs: hyperæsthesia of the legs. Absence of knee-phenomenon. Weakness of the arms. Catarrh of the bladder: death from pneumonia, within a year.

The changes in the cord and medulla were as follows:—

Posterior columns.—Degeneration of the nuclei of the funiculi graciles, and of these funiculi down to the level of the last cervical nerve, with irregular encroachment upon Burdach's columns. In the dorsal region, the disease passed over from Goll's columns to the parts just external to them, in which area it could be traced, though faintly, down to the sacral cord.

Lateral and anterior columns.—Intense degeneration of the cerebellar tracts from lumbar region to medulla. Disease of crossed

pyramidal tracts from sacral to lower cervical region, and of direct pyramidal tracts from lumbar region up to second cervical nerve.

In the upper dorsal region the disease inclined to spread diffusely over other tracts of the cord. In the gray matter, the cells of Clarke's columns had disappeared; also the median group of the anterior cornual cells: in the dorsal region indeed the whole anterior cornu was diseased.

(17.) RAYMOND. 'Arch. de Physiologie,' vol. x. 1882.

Female, æt. 78.

From age of 40, pains in the legs recurring from time to time. No other symptom till seven years before her death, when walking became difficult. She took to crutches two years later. About two years before death, after more violent pain than usual, stiffness began, first in one leg, then in the other. Soon the arms were similarly affected. While under observation, there was almost complete paralysis, with rigidity of all four limbs: exaggeration of superficial and tendon reflexes: vasomotor paralysis in the arms. There was wasting of many muscles; but their electrical reactions were normal. Death from pneumonia.

There was posterior and lateral sclerosis most marked in the cervical region. The *posterior sclerosis* was limited in the cervical and dorsal regions to the columns of Goll; in the lower dorsal region to the "Bandelettes externes," and in the lumbar region had almost disappeared. The *lateral sclerosis* was also most marked in the upper cord, and upon the left side (the left limbs had been affected before the right). It covered, according to the author's plates, a fairly well-defined triangular area in the postero-lateral columns, whose base reached the periphery [= crossed pyramidal tracts and cerebellar tracts?] The number of cells in the anterior cornua and in the columns of Clarke were diminished.

In this case, therefore, as in Strumpell's two first (Nos. 13 and 14), the spastic symptoms were predominant, and the posterior sclerosis spared the lower part of the cord.

(18.) STADELMANN. 'Deutsch. Archiv für Klinische Medicin,' vol. xxxiii. part 2, p. 125.[1]

Female, duration of disease, four years. Sacral pain, weakness of legs. Four weeks before admission, paraplegia, diminution of sensibility in the legs, paralysis of the bladder. On admission, state of hebetude, ptosis of right eye, paresis of left facial. In legs, absence of tendon-reflex, paresis of motion and sensation, pains. Arms not paralysed but hyperæsthetic.

Post-mortem. The cord was seen to be studded with small spots, the size of a pin's head, apparently hæmorrhagic.

The *posterior columns* were affected thus: in the cervical region

[1] The account given here is taken from an abstract in the 'Neurologisches Centralblatt,' 1883, p. 327.

Goll's columns were diseased; somewhat lower, Burdach's columns became involved; and lower still, the whole posterior columns were diseased. As to the *lateral columns*: in the upper cervical region there was a zone of degeneration at the periphery, which was broadest at that part where the cerebellar tract is usually placed; at the level of the cervical swelling, this peripheral degeneration was on the whole less, but still was marked over the area of the cerebellar tracts. Somewhat lower this lateral degeneration spread into the area of the crossed pyramidal tracts, which became more markedly affected still lower down. Clarke's columns were affected, their cells scanty, their ground substance cloudy.

We shall conclude this list with two cases by DÉJÉRINE. 'Archives de Physiologie,' 1884, Nov. 15th.

(19.) Male, æt. 45. Duration of disease, six years (but 15 years before he had had some symptoms of ataxy from which he had recovered). Lightning pains. Gastric, urethral, and rectal crises. Well-marked motor inco-ordination of the legs, succeeded later in the disease by paresis of the legs, without rigidity, and with absence of tendon-reflexes. Well-marked affections of sensibility in the legs, i e. patches of anæsthesia, analgesia, retarded transmission of sensations. Increase of the paresis to absolute paraplegia. Bedsore, pneumonia, and bronchitis, death.

Post-mortem.—Déjérine found degeneration of the cutaneous nerves in the anæsthetic parts, a condition which his researches in other cases have already brought into notice. There was *posterior sclerosis* distributed as follows: in the lumbar and dorsal regions over both the columns of Goll and the columns of Burdach; in the cervical region chiefly or solely over Goll's columns. The *lateral sclerosis* occupied in the lumbar region a triangular patch, its base at the periphery, its apex dipping into the cord, the disease being most intense at the periphery—in the dorsal region much the same area but reaching further forwards round the periphery of the cord—in the cervical region there was only a slight ring of sclerosis round the periphery.

There was well-marked meningitis, extending from the posterior columns round to the lateral, and corresponding in intensity to the intensity of the peripheral sclerosis.

(20.) Female, æt. 52. Lightning pains for several years; slight motor inco-ordination, with paresis, of the legs. Loss of equilibrium on closing eyes. Patches of anæsthesia and analgesia of legs. Exaggeration of tendon-reflexes: ankle-clonus. Death from broncho-pneumonia.

As in the last case, there was degeneration of the cutaneous nerves of the anæsthetic parts. In the cord there was *posterior sclerosis* through its whole length; but below the lumbar swelling, Burdach's columns were affected only in a very slight degree. (This gives some account of the exaggerated tendon-reflexes.) There was sclerosis of the periphery of the *lateral columns*, in

their posterior part, as far up as the cervical region. In Clarke's columns the cells were normal, but some of the fibres were diseased. There was meningitis, as in the last case.

The view taken by Déjérine of these two cases, (and he is inclined to extend it to the whole class we have been considering) is this,—they begin as ordinary tabes, i.e. systematic sclerosis of the posterior columns; in connection with this arises a meningitis, which spreads round to the lateral columns, and there sets up a sclerosis which is diffuse (i.e. limited to no particular tract), and spreads from the periphery inwards.

Commentary.

These cases are susceptible of some classification, and may be subdivided according to the nature of the sclerosis in the different parts of the cord. Thus both posterior and lateral sclerosis may be systematic, or the one may be systematic, the other diffuse or of doubtful nature. (We have not included cases, in which there was sclerosis, undoubtedly diffuse, of both columns;[1] though interesting cases of the kind have been published.)

In some cases, at any rate, the sclerosis of both columns appears to have been primarily systematic, and mutually independent. Kahler and Pick's first case (No. 3) is mentioned by Déjérine as the only example of this, but he appears to have overlooked Strumpell's cases (Nos. 13, 14, 15). These, with possibly some others, (Nos. 7? 10, 17,) suffice to prove the existence, as well as the mere possibility, of multiple tract disease.

It is noticeable that in some of these (Nos. 13, 14), the lateral sclerosis was more intense than the posterior, and more strictly confined to known tracts, though usually the reverse seems to hold. The posterior sclerosis was in these two cases so symmetrical and regular as to convince the author that it could not be diffuse; yet it deviated from known tracts just so far as to suggest either that our knowledge of the developmental tracts in this region is as yet incomplete,[2] or that tracts of disease may differ from the tracts of development.

Usually, however, the condition appears to have been this: posterior sclerosis presumably primary and systematic, with lateral sclerosis either diffuse or of doubtful nature. We have already noticed the explanation given by Déjérine of his own cases, viz. that the sclerosis spreads from the posterior to the lateral columns, viâ the meninges: this explanation he would extend to several of Westphal's cases, urging, with apparent reason, that the disease of the lateral columns assumed an annular form and appeared to spread inwards from the periphery. But it appears that Westphal had already considered this hypothesis and thought it inadequate,

[1] Ballet et Minor, 'Archives de Neurologie,' No. 19, abstracted in 'Brain,' No. 28. Demange, 'Revue de Médecine,' Oct. 10, 1884, abstracted in 'Brain.'

[2] The development of the tracts in the posterior columns has been studied afresh by Bechterew, 'Neurologisches Centralblatt,' Jan. 15, 1885.

for such meningeal affection as there was did not correspond in position and intensity to the lateral sclerosis. He considered the latter to be primary, though not corresponding to known developmental tracts. We must be content, therefore, to say that the nature of the lateral sclerosis is in some cases doubtful. We may remark, however, with reference to peripheral sclerosis, that it may be sometimes difficult to say whether this has spread from the meninges, or is a systematic affection of the cerebellar tracts. Perhaps the integrity or the reverse of the cells of Clarke's columns may help to determine this, as these cells are held by good authorities to be the trophic centres for the cerebellar tracts.

The histology of the lesions does not appear, as yet, to have contributed much towards settling their nature. Westphal, in some of his cases, notices a difference in the histological character of the disease in the two parts of the cord: in the posterior columns there was grey degeneration, or sclerosis, characterised by overgrowth of fibrillar interstitial tissue and disappearance of nerve elements; in the lateral columns, a degeneration characterised by the presence of numerous granular cells. But these are, in his view, only different stages of the same process. Other authors, e.g. Rabesiu, mention that the minute lesions resembled those of insular sclerosis, i.e. increase of neuroglia nuclei, presence of amyloid bodies, hypertrophy of axis cylinders, &c. But Déjérine roundly declares, that upon none of these things can we base a classification; but that the only reliable distinction between degeneration beginning in the nerve fibres and that beginning in the interstitial tissue, is to be found in their distribution; the former is systematic, the latter diffuse.

Indeed it cannot be a matter of surprise, that we know so little of the causes and of the histological beginnings of complex sclerosis, considering that our knowledge, in these respects, of the simple forms is very limited. We may begin indeed with something definite in the shape of "Wallerian degeneration," the degeneration which the fibres of a peripheral nerve undergo when separated from their nutritive centre. Comparable, if not identical with this is the process of secondary degeneration in the motor tracts of the cord when separated from their cerebral centres. But the nature of the primary degenerations is a matter of less certainty. Take the commonest of them—ordinary tabes dorsalis. Concerning this disease the commonly accepted view is that, since it originates and spreads in a district which is defined by nervous function rather than by anatomical characteristics, it must originate in the nerve-fibres. Yet, as readers of 'BRAIN' will remember, Dr. Buzzard has recently published a case,[1] in which, though the symptoms during life, confirmed by the appearances after death, pointed conclusively to tabes dorsalis, yet there was good reason to think the disease originated in the neighbourhood of the blood-vessels. This is a confirmation of the views promulgated by Adamkiewitch at the International Congress of 1881,

[1] 'BRAIN,' No. 24.

that tabes is an interstitial degeneration following the distribution of the blood-vessels in the posterior columns. But Adamkiewitch himself admits that this theory will not cover all cases, for he says in a later publication,[1] "there are two kinds of tabes, the one an interstitial degeneration originating in an overgrowth of the connective tissue which accompanies the arteries that traverse the posterior columns, the other originating from the nerves, and therefore parenchymatous in its character."[2]

Let us admit, however, what is usually believed, that as a rule systematic sclerosis originates in the nerve-fibres, and is therefore parenchymatous or "peritubular"; we have got to explain why particular tracts of fibres should be picked out by disease. This question is not yet removed from the field of conjecture. Shall we say that the tracts which in the course of development are latest to be completed, are the first to feel the effects of dissolution? Or shall we suppose that certain noxious agents, such, for instance, as syphilis, can centre themselves upon definite nerve districts, in the same way as the poisons of ergot and lathyrus appear to do?

With reference to the symptoms and the diagnosis, we will endeavour to summarise the hints that have been thrown out by the various authors we have quoted. Broadly speaking, it may be said that the symptoms due to lesion of either column manifest themselves so far as they are not neutralised by lesion of the other column. Assuming, for instance, the existence of a complete posterior sclerosis, we may recognise an additional lateral sclerosis (according to Westphal) by the addition of motor paresis to the symptoms of ataxia. In some such cases the paresis grows up side by side with the ataxia, modifying the ataxic gait from the beginning of the case; in others, according to Déjérine, the paralytic symptoms do not appear till quite a late period of the disease. But as to the other symptoms which usually accompany lateral sclerosis, viz. rigidity of the legs, exaggeration of tendon-reflex, ankle-clonus, &c., these, in the presence of well-marked posterior sclerosis affecting the lumbar region,[3] do not make their appearance, but the tendon-reflex remains absent, as in simple posterior sclerosis.

In other cases, however, the lateral sclerosis and its symptoms predominate. This is particularly the case when the posterior sclerosis (as sometimes happens) is imperfectly developed in the lumbar region. Paraplegia with rigidity then constitutes the leading feature. If the paraplegia be complete, of course no ataxia of the lower limbs can be made out. In such cases the posterior sclerosis may manifest itself either by ataxia of the upper limbs or

[1] 'Neurologisches Centralblatt,' 1884, p. 402. Report from the International Medical Congress of Copenhagen.

[2] The three cases quoted from Buzzard, Ballet & Minor, and Demange, will be found highly interesting, as illustrating "peri-vascular" sclerosis. In the two first there was a history of syphilis, in the last the vascular disease was atheromatous.

[3] Assuming, that is, that the posterior sclerosis involves the posterior root-zones; not merely the columns of Goll.

by sensory symptoms, such as lightning pains, anæsthesia, or the like, or by troubles of micturition, like those observed in ordinary tabes, or even by optic atrophy.

It deserves notice, that sometimes (Strumpell, No. 13; Raymond, No. 17) the symptoms of spastic paraplegia have been accompanied with such muscular wasting as to give the case the appearance of amyotrophic lateral sclerosis. It does not appear, however, that any reaction of degeneration has been observed.

On the whole, it seems that the elements for diagnosis of combined lateral and posterior sclerosis generally exist, and in fact the diagnosis has been more than once made.

Report.

ON RECENT ADVANCES IN THE ANATOMY OF THE NERVOUS SYSTEM.

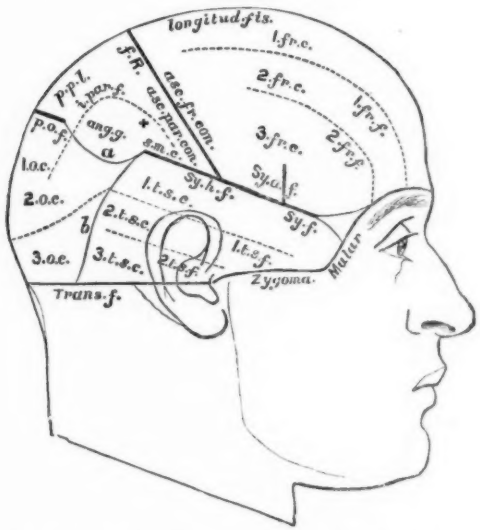
BY JAMES ANDERSON, M.D.

In the Marshall Hall Oration of last year, after speaking of the lamentable effects of Cerebral Tumours, Dr. Ferrier asked the question: "Granting that the principles of diagnosis of reachable disease shall have become established with as near an approach to certainty as is possible, where all is hidden from the eye and hand, is there any reason why a surgeon should shrink from opening the cranial cavity who fearlessly exposes the abdominal viscera?" Just one year after it was put, the question received a practical answer in the excision by Mr. Rickman Godlee of a walnut-sized glioma from the upper part of the ascending frontal convolution. The patient was under the care of Dr. Hughes Bennett, whose diagnosis of the position of the tumour was fully confirmed by the operation. Much to the disappointment of all, the patient, after a prolonged period of steady recovery, has succumbed; but while we are thus prevented from congratulating those chiefly concerned, we hope to learn useful lessons from their account of the course of the case. Doubtless there are restrictions to cerebral surgery which do not exist in the case of abdominal surgery, but we may cherish the expectation that, with the perfecting of medical diagnosis and of surgical manipulation, will come such an advance as Dr. Ferrier in his oration ventured to predict. In order to such an advance, one of the first necessities is an accurate account of the topographical relation of the various parts of the brain to the surface of the cranium. This has been already partly supplied by the work of Broca, Hefftler, Turner, and others; but the work of these observers consisted largely in laying down the relation of the cerebral convolutions to the cranial sutures, and important as that work is, it gives but little guidance either in diagnosis or operation, seeing that the sutures are but rarely perceptible through the scalp. What was wanted was the accurate laying down of the bearings of all the principal parts of the brain in relation to readily ascertained landmarks. This want has been supplied in an admirable paper published by Dr. Robert W. Reid. (*Lancet*, 27th Sept. 1884.)

The two excellent plates with which Dr. Reid illustrates his paper we are able, through his kindness, to reproduce. They largely explain themselves, and we may add, although not stated

by Dr. Reid, that they are drawn to the scale of $\frac{1}{4}$. It is to be noted as Dr. Reid says, first, that in no two brains are the sulci precisely alike; and second, that the lines and areæ on the greater surface of the scalp cannot correspond accurately with the sulci and convolutions indicated by them on the smallest surface of the brain; but granting this, we may, by choosing easily felt landmarks, so lay down the bearings of any particular point of the brain that we shall be able with almost absolute certainty to include it within some part of the area exposed by a single application of the one-inch trephine. The base line (Fig. 1) is drawn through the

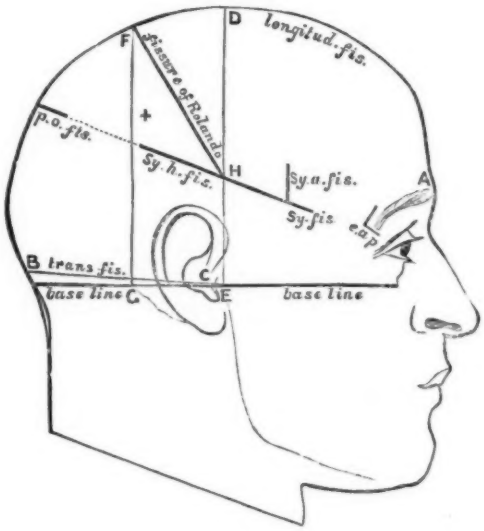
FIG. 1.



lowest part of the infra-orbital margin and the middle of the external auditory meatus, and is a definite landmark. So also are the glabella, the external occipital protuberance and the external angular process of the frontal. The most indefinite landmark used is that marked +, the most prominent part of the parietal eminence; and in many cases the posterior end of the base of the mastoid process is also very indefinite. For this reason we should prefer that the marking out of the fissure of Rolando were independent of these two points. As indicating the position of the two central convolutions it is most important that this fissure be accurately laid down, and for this purpose we prefer the method given by Dr. Arthur W. Hare in his short but most complete paper on the subject (*Journal of Anat. and Phys.* vol. xviii. Jan. 1884). Dr. Hare finds, on an average of 11 cases, that the distance from the glabella to the upper end of the fissure of Rolando is 55·7 per

cent. of the distance between the glabella and the external occipital protuberance measured along the longitudinal sinus. In no case did its variation from the point thus calculated amount to $\frac{1}{3}$ inch. Hence if the distance from the glabella to the external occipital protuberance be 11 inches, that from the glabella to the fissure of Rolando will be $6\frac{1}{10}$ inches; for $11\frac{1}{2}$ inches it will be $6\frac{2}{5}$; for 12 inches, $6\frac{3}{5}$; for $12\frac{1}{2}$ inches, 7; for 13 inches, $7\frac{1}{5}$; for $13\frac{1}{2}$ inches, $7\frac{1}{2}$. The length of the fissure, neglecting its somewhat $\int$-shaped curvature, varies from $3\frac{1}{8}$ to $3\frac{1}{2}$ inches, and it makes on an average an angle of 60° with the mesial line of the head. These two data give us the inferior extremity of the fissure, or within less than half an inch of it. Dr. Reid first marks off the fissure of Sylvius

FIG. 2.



by drawing a line from a point of $1\frac{1}{4}$ inches behind the external angular process of the frontal, to a point $\frac{3}{4}$ inch below the most prominent part of the parietal eminence. The first three-quarters of an inch of this line represents the main fissure, the rest its horizontal limb. The ascending limb starts 2 inches behind and a little above the external angular process, and runs vertically upwards for an inch. He then deduces the positions of the fissure of Rolando and the parieto-occipital fissure by the method indicated in Fig. 1. The distance between these two fissures, according to Dr. Hare's observations, varies considerably, being on an average $1\frac{7}{8}$ inches. The method of indicating the other fissures and convolutions will be at once evident from Fig. 2. As we have said, such detailed and

accurate observations as those of Dr. Reid and Dr. Hare form the necessary foundation for any advance in cerebral surgery, and will, we doubt not, be welcomed both by surgeons and physicians.

When the ordinary methods of dissection had done their best to unravel the complicated strands of the cerebral and spinal nerve-fibres, there remained yet much to be done, and to no one have we owed more in completing our knowledge than to Professor Flechsig, of Leipzig. His results have been obtained mainly by the use of two methods: first, that of embryology, and second, that of pathology. The first method consists in the observation of the different groups of fibres which acquire their medullary sheaths at the same period; the second is the marking out of tracts of fibres by their secondary (Wallerian) degeneration centrally or peripherally after section or injury, and also in some few cases by the non-development of fibres owing to the congenital absence of their centres. These results Professor Flechsig has summarised in his 'Plan of the Human Brain' (*Plan des menschlichen Gehirns;* Leipzig: Veit & Co., 1883) with its accompanying explanatory text. It will not be expected that we should criticise this plan in detail. Indeed great part of it is now beyond criticism, being incorporated among the well-ascertained facts of neurology. His account of the crusta and tegmentum of the crus cerebri is now substantially accepted, while there may still be doubt as to the exclusively cerebellar connection of the inner and outer portions of the crusta. He has also doubtless, as Professor Wernicke in a recent criticism remarks, attributed to the frontal and temporo-occipital zones a too purely mental function as opposed to the sensori-motor parietal lobe. He lays very great weight, from a physiological point of view, upon the relation of the sutures of the skull to the underlying cerebrum. This can hardly be said to be substantiated.

Alongside of Flechsig's 'Plan,' we have Professor Aeby's Scheme of the Nerve Tracts in the Human Brain and Spinal Cord' (*Schema des Faserverlaufes von menschlichen Gehirn und Rückenmark;* Bern, J. Dalp, 1884) which has already reached its second edition. It agrees in all large facts with that of Professor Flechsig, but is in many respects more complete than his and more easily intelligible. It gives both a transverse and antero-posterior section, besides subsidiary sections of the crus cerebri and the fourth ventricle. The work is accompanied by an admirable explanatory text, which closely agrees with the account of the central nervous system given by Professor Wernicke in his work on Nervous Diseases, a detailed criticism on the anatomical part of which, by Dr. James Ross, will be found in a previous volume (Brain, Oct. 1883, p. 398).

Professor Burt Wilder in his recent Cartwright Lectures at New York (*New York Medical Journal*, Feb. 9th, 1884, *et seq.*) tells us how to preserve, examine and describe the human brain. This is a subject on which we need instruction (sorely need it, in fact), for Dr. Wilder's amusing account in his second lecture of what happens

in the dissecting-room on the removal of the calvarium will doubtless recall familiar if not pleasant memories to those who read it. Our modes of preserving and studying the brain are undoubtedly crude and unsatisfactory, and Dr. Wilder's lectures, which we have read through with considerable care, contain many points which indicate that could we but spend a day with him in his laboratory, we should learn from him much that would prove practically useful. Before deriving benefit or information, however, from Dr. Wilder's lectures, the reader will find that he has a new nomenclature, in fact almost a new language, to learn. In removing the brain he will find that he is directed to make "a hemisection a little laterad of the meson," the meaning of which he may guess at, but will not be at all certain of. Again, he will scarcely recognise the familiar third ventricle as the diacele, or the cerebellum as the epen; and to be told that a new and little-known structure lies "dorso-cephalad" of the calcar, may not, without explanation, convey a definite idea of locality to his mind. As far as the actual development and comparative anatomy of the brain is concerned, we must frankly say that the reader would hardly be repaid by Dr. Wilder's lectures for the trouble of mastering his nomenclature, as he will find a more compact and intelligible account of it in Huxley's 'Anatomy of Vertebrates,' to which, along with the list of names in Quain's 'Anatomy,' Dr. Wilder acknowledges his indebtedness. Having said this, however, we can cordially recommend a perusal of Dr. Wilder's lectures, embodying as they do the detailed, persevering, and most loving study of an organ, which well repays such study even from a more or less strictly morphological view-point. He recommends that the brains of various animals should be used for practice,—those of the frog, necturus and cat, with embryonic brains, being principally used in his laboratory. For practice, it may not be so necessary in this country to follow his advice; but just as every student of medicine should more or less thoroughly dissect two or more animals besides man, in order fully to appreciate human anatomy, so specially he ought to dissect the brains of several animals. The human brain is, as regards the general animal kingdom, almost a monstrosity, owing to the huge development of the cerebral hemispheres, and its true morphology can be learnt only from lower and less complicated brains. The removal of the brain intact from the cranium is an operation requiring some skill and more than some practice. We feel tolerably certain that Dr. Wilder's method of supporting the brain in brine would in most hands result in disaster. The continuous injection with alcohol (continuous "alinjection") of the brain *in situ* has much to commend it, if the bleaching of the grey matter, which is its main disadvantage, be corrected by subsequent observation of the fresh brain. The importance attached to the description of the cerebral cavities by Dr. Wilder is a point in which he differs from our ordinary works on the anatomy of the brain, and his method of injecting the ventricular cavity from the infundibulum is devised in order to obtain a more exact impression of the shape and relation of these

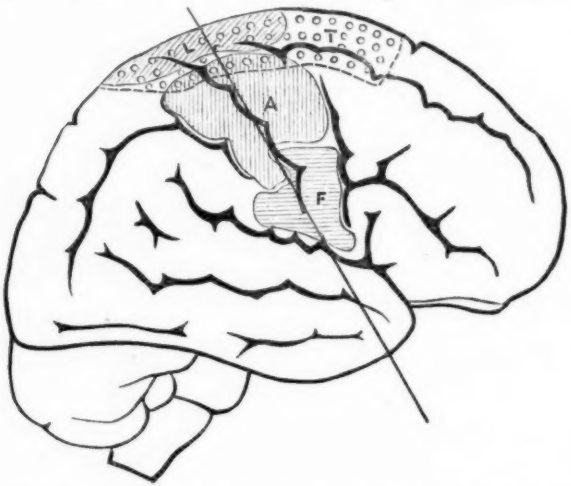
cavities. In Dr. Wilder's third and most important lecture, which we shall notice in a subsequent issue, he proposes to discuss the prevailing errors and omissions in the ordinary descriptions of the brain, along with points in cerebral anatomy still requiring elucidation.

Our knowledge of the functions of the cerebellum may be roughly summed up, by saying that it is in some way concerned in the co-ordination of movements. How it is concerned in this, and whether it has any other function, we do not know with any certainty, and neither physiological experiment, nor pathological lesions, have in recent years much advanced our knowledge. Professor Spitzka, of New York, has recently (*Alienist and Neurologist*, Jan. 1884, p. 92), by a happy reversion to a method from which we had ceased to hope for further light, attempted to solve the problem by a study of its comparative anatomy and its anatomical relations. He points out that the cerebellum steadily increases in size and complicacy as we rise in the scale of animals, becoming gradually more and more overlapped, however, by the backward-projecting cerebrum. This marked and concomitant growth of the cerebellum is a cardinal point, which we must not allow to be concealed from us by the complete overlapping of the cerebrum in the anthropoid apes and man. The vermiform process alone is represented in reptiles and birds, and in most mammals this forms a markedly separate central lobe, with its own grey nucleus, the *nucleus fastigii* in the roof of the fourth ventricle. In man this central lobe is dwarfed by the great development of the cerebellar hemispheres, with its complicated grey cortex and its grey *corpus dentatum* in the centre. The fibres connected with the cerebellum are, (1) The three crura which Professor Spitzka prefers to term tegmentibrachium, pontibrachium, and myelobrachium; (2) the trapezium, or deep transverse fibres of the pons; and (3); the auditory nerve. Of the function of the trapezium nothing definite is known; but otherwise he points out that the cerebellar connections are afferent from periphery to cerebellum, or efferent from cerebellum, or efferent from cerebellum to cerebrum; and he considers that a direct motor innervation can be entirely excluded from the functional possibilities of the organ." Through the restiform column it is connected, Professor Spitzka holds, with the posterior column of the cord, decussating by means of the olivary nucleus, and with the centripetal direct cerebellar tract. On the inner side of the restiform column is a tract which he supposes to come direct without decussation from the postero-external column of the cord. A bundle from the pneumogastric nerve has been traced to the flocculus, and Professor Spitzka has confirmed the existence of a direct cerebellar root of the sensory division of the trigeminus. The connection of the auditory nerve with the cerebellum he considers as most important in discussing the function of the cerebellum. In the lower animals, even with a rudimentary cerebellum, this auditory connection is extensive, and "with higher development the general sensory periphery and

auditory apparatus attain a more extensive projection; and this projection grows step by step with the cerebrum itself." Meynert points out that the cerebral hemispheres, the cerebellar hemispheres, the dentated nuclei of the cerebellum, and the olivary nuclei hypertrophy in proportionate degree as we proceed upward. Summing up his conclusions, Professor Spitzka says: "In other words, the cerebellum is a field where impressions of touch and position are associated with those of time and space. *Per se* this organ can do nothing, it is merely an informing depôt to the great head centre, the cerebrum. Whatever molecular oscillations are determined within its ganglionic substances must pass to higher centres for translation to skilled motor reaction."

Abstracts of British and Foreign Journals.

On Substitution. By Victor Horsley, B.S., F.R.C.S. (*Lancet*, July 5th, 1884).—After discussing the theoretical question of substitution or compensation, Mr. Horsley refers to the results of an experimental research conducted by himself and Professor



Right hemisphere of the cerebrum, showing the centres in the excito-motor area of the cortex arranged round the fissure of Rolando in the order described in the text. F. facial centre. A. Centre for upper limb. L. Centre for lower limb. T. Centre for trunk muscles. This centre being really on the mesial surface of the hemisphere, and so in the longitudinal fissure, is represented at its border by a dotted line. The straight line represents almost the plane of section shown in Fig. 2, page 81.—Cf. Fig. page 100.

Schäfer (*Proc. Royal Society*, 1884) on the subject of cerebral localisation. These observers have arrived at certain conclusions, which are modifications of previously received doctrines, as well as advanced a step in some particulars. For example, they place

the cortical centre of the lower limb at the upper extremity of the fissure of Rolando, extending backwards into the parietal lobule and forwards into the superior frontal gyrus, which is a somewhat different position from that in which it is assumed to exist by Ferrier. The list of cortical areas is completed by finding that the muscles of the trunk are regulated by centres which lie in the marginal convolution on the mesial surface of the hemisphere. They have shown that the centre for the lower limb dips largely over into the marginal convolution opposite to the upper end of the fissure of Rolando. Below these centres in order, as was before determined, come those for the upper extremity and face, and it is maintained that the fibres in the direct pyramidal tract preserve the same relative position.[1] "When opposite the centre of the lenticular nucleus the fibres become bent in entering the area of the internal capsule, and being twisted like the rays of a fan close to its hinge, now become arranged in an antero-posterior manner, the most anterior being what was before the most inferior." A hæmorrhage of the lenticulo-striate artery, which courses round the under surface of the lenticular nucleus passing through it and the internal capsule to the caudate nucleus, will destroy the pyramidal fibres in the order of face, upper limb, lower limb, and trunk, as the focus of disease will be below and in front of the bundles of fibres. This is exactly the order which is observed to be affected clinically. This arrangement of fibres from the cortical centres in the internal capsule is strikingly exemplified by a case in this number of 'BRAIN,' in which a flat limited softening in this tract caused complete paralysis of the upper extremity without any involvement of the face or leg.

Further it will be seen that Mr. Horsley's explanation accounts for the classical mode of recovery of power in hemiplegia, without resorting to the well-known theory of substitution advanced by Dr. Broadbent; the two factors being: 1. Removal of pressure by absorption of extravasated blood, &c.; 2. Further development of function in the bilaterally acting cortical centres.

Sharkey and Lawford on Acute Optic Neuritis, associated with Acute Myelitis. (*Ophthalmological Society's Transactions*, vol. iv.)—A girl, aged 17, previously in good health, rapidly lost her sight, so that in four days she was quite blind, without other prominent symptoms. Well-marked double optic neuritis was

[1] See Figures on pages 81 and 82 of the present number of 'BRAIN.

found to be present a month afterwards. Symptoms of paralysis and loss of sensation in the lower extremities supervened. About three weeks afterwards the patient died from symptoms of peritonitis; that is sixty-two days from the time vision first failed, and twenty-nine days after the first appearance of symptoms of paralysis. A very careful account of the post-mortem examination is given in detail, showing the brain to be healthy. The spinal cord presented the appearance of acute myelitis at the lower cervical and upper lumbar regions. The optic tracts and nerves, along with the discs and retinæ, were accompanied by all the evidences of intense inflammatory change.

It is pointed out that the interest in this case lies in the association of an acute optic neuritis with acute inflammation of the spinal cord. There is probably some relation between the two, although the one does not depend directly on the other. In this case the optic neuritis appeared a month before the spinal symptoms. Post-mortem examination showed that the spinal cord between the lower cervical and lumbar regions was healthy, so that the centres of disease must have originated independently one of the other. The same may be probably asserted with regard to the optic nerves. Other cases have been published, by Clifford, Allbut, Seguin, Noyes, Steffen, and Erb, in which spinal symptoms were associated with changes in the optic discs. In the *Archives of Ophthalmology*, for 1882, No. II., a case is recorded by Julian J. Chisholm, M.D., somewhat similar to the one under consideration, but without post-mortem examination. In the *Lancet* for 1882, Dr. Dreschfeld published two cases, each with an autopsy, of acute cord disease, accompanied with optic neuritis.

These cases moreover prove, that the occurrence of double optic neuritis in association with nervous disturbance is not of necessity due to cerebral disease.

Sharkey on Embolism of the right middle Cerebral Artery, producing left Hemiplegia and Hemianæsthesia, Absorption of a large portion of the Right Hemisphere: Death seven years later. (*Transactions of Medico-Chirurgical Society*, vol. lxvii. 1884.)—A woman, aged 34, with heart disease, was seven years ago seized with an apoplectic attack, followed by left hemiplegia and hemianæsthesia, including amblyopia of the left eye and deafness of the left ear. She gradually improved, and in about four months, although the arm remained almost useless, very fair power had returned in the leg, and sight and hearing were completely

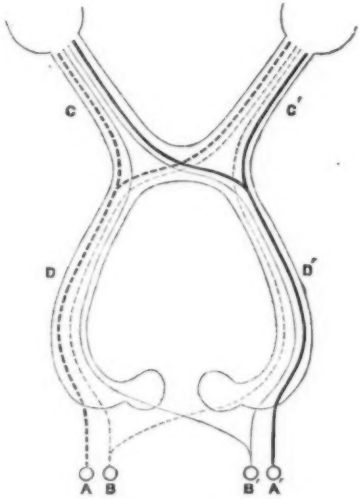
restored. Seven years afterwards she again came under observation. During this interval she had been somewhat weak-minded, but remained, according to her husband, a good-tempered, hard-working, and affectionate wife. On examination, there was weakness and rigidity of the left upper extremity, and paresis of the left leg and side of the face. The sensibility was everywhere normal; hearing and sight were as in health. The patient died suddenly. After death, in addition to heart, liver, and kidney disease, the brain was found extensively altered. The morbid condition may be summed up by saying that in the right hemisphere there was great deficiency in its substance. The convolutions affected were those supplied by the Sylvian artery, viz. the inferior frontal, the external portion of the orbital, the lower half of the two central convolutions, the inferior parietal lobule, the convolutions of the island of Reil, the angular gyrus, the inferior and middle temporo-sphenoidal, and part of the inferior occipito-temporal convolutions. These convolutions had completely disappeared, and a depressed, ill-defined, loose mass of a yellow colour remained. The right optic thalamus, lenticular nucleus, both corpora quadrigemina, and the right half of the pons, and right pyramid, were reduced in size. Drawings are given of the anatomical appearances.

The author points out that this case is one of embolism of the right middle central artery, occurring seven years before death, and resulting in subsequent absorption of those parts of the brain which are supplied by it. The chief features of interest are, that although so large a portion of one cerebral hemisphere was absent, the patient remained a useful member of society for seven years. Again, although at first there was total hemiplegia and hemianæsthesia, the loss of motion alone was permanent. Owing to the great destruction of the basal ganglia, the case is useless for purposes of cortical localisation. Still some important conclusions may be drawn. 1. Destruction of the angular gyrus and superior temporo-sphenoidal convolutions, on one side, does not involve permanent loss of sight and hearing on the other. 2. Each hemisphere is specially connected with the sight of the opposite eye. 3. Sensation on each side of the body is not so indissolubly connected with certain definite areas of the opposite hemisphere as is the case with motion.

Sharkey on Homonymous Hemianopia, probably due to a Cortical Lesion. Dr. Sharkey (*Ophthalmological Society's Transac-*

tions, vol. iv.) relates the case of a woman, aged 51, who had suffered from paroxysmal seizures for two-and-a-half years. These consisted of a play of colours in the right eye, followed by convulsion of the right hand and arm, subsequent rigidity of the leg on the same side, and finally loss of consciousness. After the original attack there was paresis of the right arm, and inability to see on the right side, without optic neuritis or other special nervous symptoms. Details of the case are given, and among others the fact, that the defect of vision did not reach as far as the vertical line drawn through the fixation point, but occupied only the peripheral field of vision on the same side in both eyes. There was no post-mortem examination, but the conclusion drawn from all the circumstances of the case was that the lesion existed in the left hemisphere, affecting the cortical centre of the arm and its neighbourhood. Immediately posterior to these are the angular gyrus and occipital lobe, both which are supposed to contain the central elements of the sense of sight. Hence the same disease might easily involve both these motor and sensory areas. Both physiological experiment and pathological observation point decisively to the view, that each hemisphere is connected with both eyes, and that there is to some extent a separation between the area in the cerebral cortex which receives the peripheral fibres of the corresponding halves of the retinæ, and the area which receives the centre fibres of the opposite retina. The course which link the fibres of the retina to the cerebral cortex have been best demonstrated by an appeal to pathological observations. It has been for some time known that diseases of the optic nerve caused complete blindness of the same eye, and that destruction of the optic tract caused homonymous hemianopia, in which the dividing line passes through the fixation point. The case under notice, however, shows that disease of some portion of the cerebral cortex produces homonymous hemianopia confined to the peripheral portions of the retinæ, their central parts retaining clear vision. Hence the fibres of the optic tracts as they pass into the hemispheres must separate in some such way that those which come from the periphery of the retina enter the cortex at a point which is, at least to some extent, distinct from that which receives the central fibres. Pathological and experimental observations moreover prove, that each hemisphere has an essential and well-marked special connection with the opposite eye; and as separate areæ in both hemispheres supply the peripheral fibres of each eye, this special

connection can only take place by means of the central fibres. Now lesions of the optic tracts prove, that the central fibres of each retina are contained partly in the tract of the corresponding side, partly in that of the opposite side. Therefore those in the tract of the same side must cross beyond the corpora geniculata, in order to reach the cortex of the hemisphere opposite to the eye



from which they come. The accompanying diagram, which is a modification of Charcot's, is given to graphically represent this reasoning. The case under consideration supports the view, that in homonymous hemianopia in which central vision is retained for some degree on all sides of the point of fixion, the cause is probably cortical.

A. Hughes Bennett.

Disseminated Sclerosis and Infectious Diseases. (P. Marie, *Progr. Médical, Apr.* 12, 1884, *et seq.*)—M. Marie opens his paper with the following case. Two years after typhoid fever, certain nervous symptoms made their appearance; these were, slow and hesitating speech, not scanned. Tremors, nystagmus, homonymous diplopia. Staggering gait. Some paresis of limbs. Increased knee-jerk, no loss of sensation. He regards these nervous symptoms as indicating the pathological condition known as dis-

seminated sclerosis, and he considers that the typhoid fever is the cause of them.

In support of this view, he quotes a considerable number of cases recorded by competent observers. Of these, 10 cases are given in which the nervous symptoms appear after typhoid fever; 4 after variola; 2 after erysipelas; 3 after pneumonia. He also gives cases occurring after measles, scarlatina, hooping-cough, intermittent fever, diphtheria, cholera, syphilis. In all these cases the prominent symptoms were tremor, paresis of limbs, and, less often, troubles of speech and nystagmus.

In only three cases (Ebstein, Jolly, Joffroy) was the diagnosis confirmed by autopsy.

In five cases the symptoms abated, or completely disappeared.

The author ends with some remarks on the pathology of disseminated sclerosis. He starts by postulating the vascular nature of the disease, and he remarks that arteritis bears a close relation to infectious diseases, and also tends to be disseminated over the body in patches. Popoff has described an accumulation of lymph-cells in the perivascular lymphatics in typhoid fever. Ribbert believes that an irritant substance (microbe) can be carried by the circulation, and being arrested by the vascular wall, determines a perivascular inflammation, which is the cause of a patch of sclerosis.

M. Marie arrives at the conclusion, that typhoid fever and other infectious diseases have a series of secondary and tertiary accidents, comparable to those of syphilis. As one of these he regards disseminated sclerosis. He suggests that what are now called "*late complications*" of the diseases, should be called more properly "*late manifestations.*"

M. Marie's paper is a very interesting one, but we must admit that, however much we may be inclined to agree with his conclusions, yet we cannot regard his premisses as particularly strong. It is much to be regretted that more autopsies are not recorded. We should decidedly hesitate to diagnose disseminated sclerosis from the meagre symptoms in some of the quoted cases.

However we cannot but hail with interest any observations on the ætiology of so obscure a disease as disseminated sclerosis, and M. Marie's remarks on the pathology of the disease are particularly instructive. H. H. Tooth.

Case of Recurrent Dropsy of the left middle Ear, complicated after eight years duration by acute attack of Monocular Optic Neuritis on the same side, followed by General Tabetic

Symptoms. By CHARLES H. BURNETT, M.D., and CHARLES A. OLIVER, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—At 8 years of age the patient had a polypus removed from the right ear; when seen at the age of 55 he was deaf with this ear, and it appeared that the canal had become closed by the growth and organisation of granulation tissue formed after the removal of the polypi. He applied, however, on account of recent deafness in the other (left) ear. The membrane was opaque and the malleus retracted. But from statements of the patient it was surmised that there was fluid in the tympanic cavity: paracentesis evacuated a little tea-coloured fluid, and gave much relief to the symptoms.

Deafness, however, recurred, and the operation was repeated with relief to this symptom as many as 38 times in the course of the next two years. At last the fluid did not return, but ordinary hypertrophic catarrh of the middle ear set in. He had by this time developed dizziness, and staggered on turning round. This symptom, however, the authors regard as "tabetic in origin" rather than as referable to the ear-disease.

About a year after the aural treatment was begun, he complained of dimness of vision in the left eye. In this (the left eye) there was well-marked optic neuritis with swelling of the disc. Vision for light only. In the right eye the optic disc was dirty grey in colour, but there was no evidence in the authors' opinion of past neuritis: there was uniform contraction of the colour fields. The optic neuritis of the left eye gradually disappeared; the disc finally assuming the dirty-grey colour seen in the right disc, and correspondingly the vision for form and colour reappeared, to disappear again as atrophy set in. In brief, simple atrophy was observed in the right optic nerve, optic neuritis with consecutive atrophy in the left.

The remaining symptoms observed were, loss of smell in the left nostril; unsteadiness when walking, exaggeration of patellar tendon-reflexes, especially on the left side; which subsequently became diminished, though the left remained always in excess of the right. The pupils acted normally to light. There was slight paresis in the right facial district, probably to be connected with the old ear-disease on that side.

The condition of the ears the authors suppose to be purely local. The uniocular neuritis and the loss of smell they ascribe either to a cerebral tumour of uncertain position or preferably to a chronic pachymeningitis at the base of the left side of the brain; the remaining symptoms to sclerosis of the posterior columns. (But

seeing that the tendon-reflexes were exaggerated, the pupils acted normally to light, and that there were no pains, the diagnosis of posterior sclerosis seems rather uncertain.)

On the neglect of Ear-symptoms in the Diagnosis of Diseases of the Nervous System. By G. L. Walton, M.D. (*Journal of Nervous and Mental Disease*, October 1883).—The author draws attention to the fact, that while the relations of the eye and its functions to nervous disease have been minutely studied, and the condition of this sensory organ is reported in all careful accounts of cerebral disease, yet the ear and the sense of hearing have been for the most part neglected.

Hysterical deafness, with loss of hearing power for high tones, analogous to hysterical blindness with contraction of the visual field and colour-blindness, is, he thinks, an example that ophthalmic facts, important from a neurological point of view, may be paralleled by aural facts. But, as a matter of fact, in the reports of nervous cases the hearing power is usually either not inquired into at all, or else any existing deafness is at once set down to the nervous disease without investigation of the peripheral organs of hearing. Thus, the seeming rarity of deafness in disease of the pons and cerebellum is probably due to the fact that the hearing is not systematically examined in such cases. Actual examination is necessary, for the patient may be quite ignorant that he is deaf, and deafness of one ear may evade the physician's notice if not looked for. But even where deafness is reported, there is often either no examination of the ears made, or a most superficial one. Thus, cases of locomotor ataxy with deafness are frequently related; the inference being that the deafness is due to the nervous disease; whereas, according to the author's experience, it is much more often peripheral than central. The same with the so-called Ménière's disease, which is more often than not due to disease of the middle ear.

Examination of the Spinal Cord in a Case of Polio-Myelitis of the Adult of two months standing. By James J. Putman, M.D. (*Journal of Nervous and Mental Disease*, January 1883).—A single woman, æt. 22, had caught cold while menstruating, three weeks before admission; the flow was checked, and pain in the head, neck, back, and limbs set in, with vomiting. In three days there appeared paralysis of the limbs, with numbness, and inability to hold her urine.

When first examined, the temperature was 99·8°; there was tenderness along the spine, pain on movement, atrophy of the muscles of the limbs, specially marked in the right arm and intrinsic muscles of the right hand; some contracture of the right arm and hand. There was some improvement for a month; then a rise of temperature, up to 102°. After another month there came a similar rise of temperature, accompanied by vomiting and diarrhœa, which proved fatal.

The post-mortem appearances (naked eye and microscopic) are thus summarised by the author. Extensive ulcerations in the large intestine; subpleural hæmorrhages. Throughout the whole length of the spinal cord anterior and posterior poliomyelitis [each anterior and posterior cornu was threaded in its whole length by a column of inflamed tissue]; but the right anterior cornu had suffered most severely; here, in many sections, there were no healthy ganglion cells to be seen; also atrophy of the anterior nerve-roots and to a less extent of the posterior; subacute inflammation of the antero-lateral white columns; a moderate amount of lepto-meningitis; thickening of the vessels everywhere even in the posterior columns, and diffuse though moderate increase of the connective tissue.

The author suggests that diffuse myelitis would be the fittest name to apply to the case.

The Pathological Anatomy of the Cerebro-Spinal Axis of a Case of Chronic Myelitis of nineteen years standing. By Dr. H. D. Schmidt (*Journal of Nervous and Mental Disease*, July 1883).—The case originated in a gunshot-wound of the neck. At the time of the post-mortem there was no trace left of fracture of the vertebræ, or of direct injury to the cord in this situation. Complete paralysis of all four extremities succeeded the injury immediately. The paralysis of the legs disappeared, leaving the arms paralysed, and at first hyperæsthetic, subsequently anæsthetic. The arms became contractured in a year. Sixteen years after the injury the patient could walk, but there was contracture of the left foot, and commencing pains and hyperæsthesia of the lower limbs. After 18 years, muscular atrophy had begun in the hands and arms. He finally became unable to walk, lost control over his evacuations, and died in a state of coma and complete paralysis.

The macroscopic appearance of the brain was normal. The cord in the cervical region was flattened and softened, there was here a tubular cavity apparently caused by the softening.

Further, a yellowish-grey discoloration was observed in the posterior columns, in the left postero-lateral column and in the periphery of the white substance, and in spots scattered through the rest of the white substance. It diminished from above downwards, and does not appear to have been strictly systematised; it was generally diffused through the pons and medulla. Microscopically, the following points were made out. (α) There was degeneration of almost all the vessels of the cord—the arteries of the pia mater (which were least affected) showed thickening of the adventitia and indistinctness of the muscular coat, while the walls of the smaller vessels within the cord and medulla were pale, granular and indistinct. (β) Round the vessels, both upon the large and small septa of the cord, was a fibrinous exudate, pale and granular, but staining deeply with carmine. (γ) There was atrophy of nerve fibres very variable in degree, apparently caused by pressure from the exuded material. (δ) Numerous rounded bodies were observed, both in the grey and white matter, specially in the medulla and pons. These could not be proved chemically to consist either of fat or of amyloid substance. The author considers them to have been ganglion cells of the grey matter and nerve-nuclei of the white substance in various degrees of degeneration.

Two Cases of compression of the Spinal Cord by Sarcomatous growths from the Soft Membranes. By G. Long Fox, M.D., F.R.C.P. (*Bristol Medico-Chirurgical Journal*, July, 1883).—*Case* 1.—A woman æt. 35, strained herself while lifting a window, and a month after began to feel pain in the right arm and weakness in the right hand. Some months later, after getting wet, loss of power in both legs and arms was noticed. When seen nine or ten months after the original strain, there was almost complete motor paralysis of both arms, sensation being also impaired, though not absent; and complete motor paralysis, with dulness of sensation, in the legs. There had been incontinence of urine and constipation. Pupils regular. Tendon-reactions exaggerated. No distinct muscular atrophy. Some tenderness over 6th cervical vertebra (a seton had been worn there). Death in five months more, from bronchitis and paralysis of the intercostals.

Post-mortem, a hard rather vascular tumour (having the microscopic characters of a spindle-celled sarcoma), measuring $1\frac{1}{2} \times \frac{3}{4}$ in. was found growing from the arachnoid on the anterior surface of

the cord in the cervical region. It had there destroyed by pressure all the cord except the posterior columns. The cord was almost deliquescent at the level of the tumour, and softened down to the lumbar region.

Case 2.—Shooting-pains in the arms, especially at night, for three months, the right arm being worst: then loss of power of extension, first in the right fingers, and next in the left. For five weeks, numbness and pricking in the toes, first of the right foot, and then of the left; gradually this extended upwards to the waist. Priapism and difficulty in micturition. When examined, there was anæsthesia up to the third rib, and paralysis of the lower limbs. Plantar reflexes normal. The fingers were at first the only parts of the upper limbs that were paralysed, but gradually nearly the whole arms became involved. Thoracic respiratory movements began to fail before death.

Post-mortem.—Well-defined tumour (spindle-celled sarcoma), measuring $1\frac{1}{2}$ inch × $\frac{3}{4}$ inch, on the left posterior aspect of cord in cervical region. Much softening of the cord in its neighbourhood. Along the whole length of the cord below was a belt of yellowish-grey substance $\frac{1}{4}$ inch thick, enveloping the cord on all sides equally.

Seguin on the American method of giving Potassium Iodide in very large doses. (*Arch. of Medicine*, 1884, p. 114.)—The author alludes more particularly to the later nervous lesions of syphilis. In many cases he admits that ordinary doses (viz. up to 8 grammes, or 120 grains daily) are sufficient. But it often happens that 10 or 15 grammes a day have to be administered, and this dose increased week by week, before results can be obtained. For instance, in syphilitic headache Seguin prescribes two doses of 4 grammes (60 grains) each on the first day, and increases this quantity by one dose every day until 32 grammes (1 ounce) are taken in the twenty-four hours. Very large quantities must be taken in syphilitic coma, especially when convulsions or choked-disc are present. It is obvious that in specific hemiplegia, and the like, the iodide cannot be expected to remove the symptoms depending upon actual destruction of nervous tissue. The salt should always be given on an empty stomach, largely diluted with some alkaline water.

On the efficacy of Iodide of Potassium in non-Syphilitic Organic Diseases of the Nervous System. By Dr. E. C. Seguin.—The author gives three classes of cases.

I. In which symptoms were temporarily relieved, and in which, post-mortem, the lesion was proved to be non-syphilitic.

Case 1.—Right-sided hemiparesis with inco-ordination, palsy of left external rectus; headache, vomiting, optic neuritis. Improvement under iodide of potassium (10 to 40 drops of a saturated solution three times daily for two months). Death about five years afterwards: *sarcoma of left crus cerebri.*

Case 2.—Headache, vomiting, staggering gait; exophthalmos, double optic neuritis; a soft pulsating tumour in region of lambdoid suture. Iodide from 90 to 150 grains per diem; some relief to symptoms. Death within a few months: *fibro-sarcoma of cerebellum, with dropsy of the ventricles.*

Case 3.—Fall on back of head; subsequently attacks of vomiting with headache: paralysis, especially of the left side, optic atrophy. Four years later, right hemiparesis, absence of tendon-reflex at knees, epileptiform attacks with occipital pain. Iodide (10 to 40 drops of a saturated solution), taken three times daily, cut short the fits. Death next year. *Sarcoma of right hemisphere of cerebellum; meningitis of convexity of cerebrum.*

II. Cases without post-mortem; symptoms cured or relieved by iodide, no evidence of syphilis.

Case 1.—Numbness of left side, mental affection. Numbness disappeared while taking iodide; patient lapsed into dementia paralytica.

Case 2.—Paralysis of third nerves; paresis and ataxia of limbs. Two attacks were relieved by iodide of potassium, 30 to 120 drops of the saturated solution three times daily. A third attack appears to have resisted all treatment.

Case 3.—Right-sided convulsions affecting the face principally; aphasia. Recovery while taking iodide of potassium, at first combined with the bromide.

III. Cases of double optic neuritis in children, probably due to basal meningitis; apparent good results from large doses of the iodide. Three cases are given; headache, vomiting, and internal squint were observed in addition to the optic neuritis.

The author gives the iodide largely diluted with water, Vichy water, or solution of bicarbonate of soda.

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